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BULLETIN OF
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THE VELOCITY OF CONDUCTION IN NERVE FIBER AND ITS ELECTRIC CHARACTERISTICS

I. OPATOWSKI
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

The theory developed in this paper shows that the propagation of spike potential along a nerve fiber and the conduction of an electric wave along an inert inorganic conductor follow a common quantitative relationship. This result gives further support to the belief that propagation of excitation is an electrical process. The basic idea of the theory is derived from the consideration that velocity has, by its mathematical definition, a local meaning; conduction in a nerve is completely determined by the local characteristics of the latter, as well as those of the wave. The final formula derived does not make use of any other field of science beyond the fundamental principles of electricity. It gives the conduction velocity in terms of the electric characteristics of the fiber and of the duration of the spike potential. The formula is in agreement with the known dependence of the conduction velocity on various parameters characterizing the axon. The computed velocity agrees with the measured ones on the squid giant axon, crab nerve axon, frog muscle fiber and Nitella cell. The membrane inductance appears as a velocity controling agent which prevents also a possible distortion of the spike potential during conduction. The structural meaning of the electric characteristics of the axon membrane is discussed from the viewpoint of the diffusion theory. A formula for the velocity of spread of the electrotonus is also derived.

The object of the present paper is: (i) to derive a formula for the velocity of propagation of excitation in terms of the electric characteristics of the axon and of the duration of the spike potential; (ii) to discuss a possible structural meaning of the known data on the electrical properties of the axon; (iii) to examine the velocity of spread of the subthreshold potential from an electrical viewpoint.

1. *Axon as an Electric Circuit.*

We base our theory on the following well-known electric circuit representing a nerve fiber (Fig. 1) (see, e.g., Cole and Baker, 1941; Curtis and Cole, 1944; Hodgkin and Rushton, 1946; Davis and Lorente de Nò, 1947). The axoplasm and the external medium are considered as distributed longitudinal resistances of r_I and r_E ohm/cm respectively, in parallel with respect to each other. These resistances

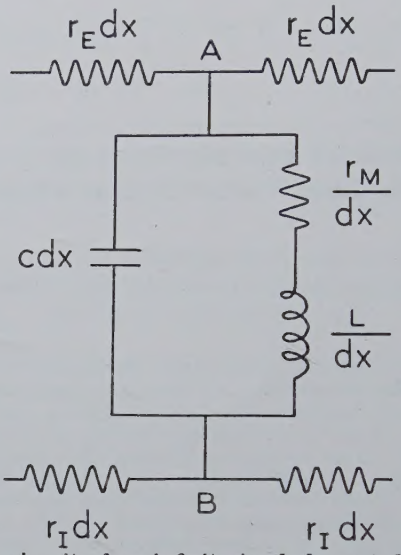

FIGURE 1. Electric circuit of an infinitesimal element dx of a fiber.

are connected transversally by a uniformly distributed membrane impedance consisting of a capacity of c farad/cm in parallel with a resistance of r_M ohm cm and with an inductance of L henry cm, both per unit length of the fiber and both in series with each other. Although inductance is considered in the formulas, we do not exclude the case $L = 0$; some of our conclusions are practically independent of L.

2. *Wave propagation in an Electric Conductor and Conduction of Excitation in an Axon.*

The theory of electricity offers a simple mathematical description of propagation of a wave caused by a harmonic electromotive force applied at one end of a semi-infinite conductor (see, e.g., Pender and Warren, 1943, pp. 201–09). In various theories axons have often been considered to be of infinite length (Rashevsky, 1948; Hodgkin and Rushton, 1946; Davis and Lorente de Nò, 1947) and this approximation seems to be quite good under appropriate experimental conditions. The type of conductor that is usually employed in electrical engineering is somewhat different from the circuit represented in Figure 1. This is mainly because in transmission lines the inductance is in series with one of the longitudinal conductors and not in parallel with the capacity (see, e.g., Knowlton, 1949, pp. 1184-86, 2047-49; Pender and Warren, *loc. cit.*). For this reason we have to derive first of all a formula for the propagation of a

simple harmonic perturbation in a conductor such as that represented in Figure 1. We consider the steady state and will justify at the end of this section, as well as in the following sections, the procedure of considering the conduction of excitation as a propagation of the simple harmonic perturbation in the steady state. We use the method of complex variable of the theory of alternating currents (see, e.g., Pender and Warren, 1943, chap. vi). If V is the complex voltage across the membrane, that is, between A and B (Fig. 1), we have, in the steady state,

$$d^2V/dx^2 = k^2V \quad \text{with} \quad k^2 = (r_E + r_I)/z\,, \tag{1}$$

where z is the complex impedance of the membrane, per unit length:

$$1/z = r_M(r_M^2 + \omega^2L^2) + j\omega\,[c - L(r_M^2 + \omega^2L^2)^{-1}] \tag{2}$$

with $j = \sqrt{-1}$ and $\omega = 2\pi/T$, the period of the voltage considered being T sec. Equation (1) can be obtained from a formula which has been used in the case of direct current [Cole and Hodgkin, 1939, eq. (8)] by substituting for the membrane resistance r_M its complex impedance z. Equation (2) is simply an expression of the fact that c is in parallel with r_M and L (see Fig. 1). We put $V(+\infty) = 0$, $V(0) = V_0$. Then the solution of equation (1) is:

$$V(x) = V_0\, e^{-\alpha x}(\cos\beta x - j \sin\beta x), \text{ where } k = \alpha + j\beta\,, \tag{3}$$

α and β being two positive quantities and x the distance measured along the axis of the conductor. Since $k^2 = \alpha^2 - \beta^2 + 2j\,\alpha\,\beta$, from expression (1) of k^2 and from relation (2), a system of two equations in α and β is obtained, which, putting

$$S = \left[\left(\frac{r_M}{r_M^2 + \omega^2L^2}\right)^2 + \left(\omega c - \frac{\omega L}{r_M^2 + \omega^2L^2}\right)^2\right]^{1/2}, \tag{4}$$

gives

$$\beta = \left[\frac{r_E + r_I}{2}\left(S - \frac{r_M}{r_M^2 + \omega^2L^2}\right)\right]^{1/2}. \tag{5}$$

The quantity α is given by the same expression as β except that a plus sign appears in the parenthesis, after S, instead of a minus sign.

Since we consider a simple sinusoidal electromotive force at $x = 0$, we can write for the instantaneous value of the voltage V_0:

$$|V_0| \sin \omega t\,. \tag{6}$$

Of course, the assumption of a harmonic electromotive force at $x = 0$ should not be interpreted as an analogue of a similar electro-

motive force actually acting in an axon. This electromotive force is introduced here only for the purpose of deriving a simple formula for the velocity of conduction of a perturbation in a conductor whose elementary electrical characteristics are the same as those of an axon (Fig. 1). The reasons why this formula is applicable to the velocity of propagation of excitation will be given later.

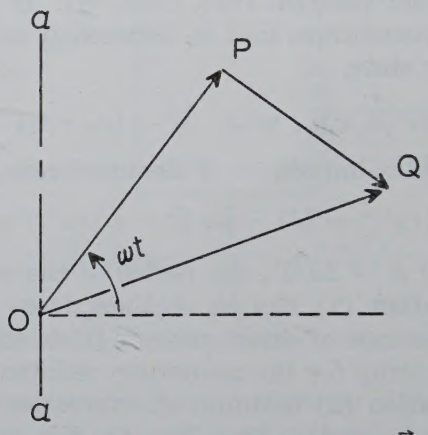

FIGURE 2. Vector diagram of equation (3). $\vec{OP}$ $|V_0|\ e^{-\alpha x} \cos \beta x$, $\vec{PQ} = |V_0|\ e^{-\alpha x} \sin \beta x$. The instantaneous value of the voltage V is given according to equation (3) by the projection of OQ on aa.

Relations (3) and (6) can be represented by the vector diagram of Figure 2 in which all the vectors rotate around O with an angular speed of ω radians per second and the instantaneous value of the voltage V is given by the projection of $\vec{OQ}$ on the axis aa. This is a known representation of sinusoidal quantities by rotating vectors. Use is made here of the fact that multiplication of a vector by $-j$ means its rotation by $-90°$. Consequently, using relations (3) and (6) we see the instantaneous value of the voltage V at a moment t and point x is (cf. Fig. 2):

$$|V_0|\ e^{-\alpha x} \sin(\omega t - \beta x). \tag{7}$$

The coefficient α represents the damping of the wave owing to dissipation of energy in the resistances. If we neglect the damping, expression (7) has the form of a wave propagating with a constant speed equal to:

$$v = \omega/\beta = 2\pi/\beta T. \tag{8}$$

The main objective of the present paper is to show that *formula*

(8) *expresses* with good approximation *the velocity of conduction of the spike potential in an axon, where* T *is the duration of the spike.* One of the first questions which presents itself in this connection is the following: *How can a formula for the velocity of propagation of a sinusoidal wave in an inert conductor be used to calculate the velocity of conduction of a spike potential in an active axon and why can the damping be neglected in this calculation?*

To answer this, let us examine first the physical conditions determining the velocity of conduction of sinusoidal wave in a conductor. The function V which we have considered represents the potential difference between A and B (Fig. 1). However, except for a proportionality factor and possibly also for a different reference potential as zero potential, V is also the potential in the extrapolar regions of the external resistance [Davis and Lorente de Nò, 1947, formula (14); Hodgkin and Rushton, 1946, formula (1.4); Hodgkin, 1947b, p. 324]. Therefore, except for terms which do not affect the wave character of expression (7), the latter represents not only the voltage across AB but also the potential in the extrapolar regions of the external medium. Figure 3 gives the diagram of this poten-

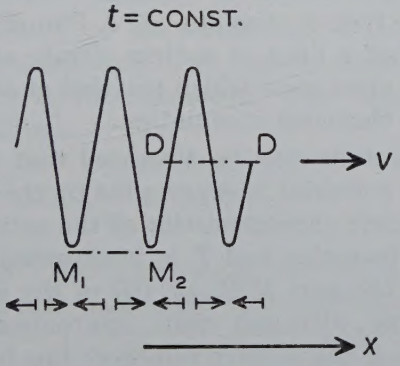

FIGURE 3. Diagram of a sinusoidal voltage wave as an analogue of a spike potential, monophasic if the reference line is taken in M_1M_2, diphasic if it is taken in DD. The arrows below the curve give the directions of the local currents.

tial at a fixed moment t in the case of an inactive conductor (represented by Fig. 1) which has a negligible damping. This diagram represents a wave moving at a constant speed v. The arrows below the curve represent the directions of the local currents in the external medium at the moment t considered. For simplicity, these arrows are drawn on the assumption that the phase difference between

current and voltage is negligible; the directions of the arrows express then the elementary fact that current flows in the direction of decreasing potential. Let us consider one wave length, for instance, that lying in M_1M_2 (Fig. 3). What are the quantities on which the rate of advancement of this wave length depends in a direct manner? Since velocity involves distance and time up to infinitesimals of first order only ($v = dx/dt$), the rate of advancement of the part of wave M_1M_2 at the moment t is determined completely by the conditions of the conductor from $M_1 - dx$ to $M_2 + dx$ during the time from t to $t + dt$. These conditions are expressed by the values of the electric constants which that part of the conductor has during that time. Conditions of the parts of the conductor which between the moments t and $t + dt$ are distant from M_1M_2 by more than infinitesimals of first order in x do not affect the velocity of the part M_1M_2 of the wave at the moment t. (For other applications of this type of argument to the velocity of conduction in nerve see Opatowski, 1950, 1951). One could think a priori that the velocity of propagation of the wave might depend on the magnitude of the voltage. Formulas (8) and (5) show that this is not the case. In the physiological field there are indications that conduction velocity does not depend even on the type of stimulus. R. J. Pumphrey and J. Z. Young (1938) mention that a drop of sodium citrate solution produced an impulse in a squid giant axon which traveled at about the same speed as one induced by electrical stimulation.

Now the conjecture may be advanced that the velocity of conduction of a spike potential is expressible by the same formulas (8) and (5) if the electric characteristics of the active part of the axon are used in these formulas and T is interpreted as the duration of the spike. In fact the part M_1M_2 or DD of the wave shown in Figure 3 is quite a good, although crude, approximation of a spike potential, monophasic if the voltage reference line is taken as in M_1M_2, diphasic if it is taken as in DD. The fact that in an axon a single piece of wave, for instance, M_1M_2, appears whereas in Figure 3, which represents an electrical phenomenon in a purely inert conductor, that piece is preceded and followed by many other identical wave pieces is immaterial as far as the velocity of conduction of M_1M_2 is concerned. This velocity is independent of what happens beyond M_1M_2 except for infinitesimals of first order, as already explained. *Thus what happens within one wave length of a conductor through which alternating current flows is a good formal analogue of what happens in an active part of the axon.* As a matter of fact the direc-

tions of the currents drawn in Figure 3 for an ordinary conductor well represent the local action currents in the external medium of the axon, if M_1M_2, for instance, is taken as the spike potential (cf. e.g., Lloyd, 1947, p. 11). *Thus the local action currents do not appear to be a purely biological phenomenon, since they exist in their essential characteristics also in inert conductors during wave propagation.*

As to the reasons for considering damping to be negligible, although dissipation of energy must occur in electric resistances, one justification only seems to be possible. The internal energies which are believed to be released during excitation (cf. e.g., Curtis and Cole, 1944) compensate for the ohmic loss of energy in the axon.

Reasoning which has lead us so far to conclude that formulas (8) and (5) should express the velocity of conduction of a spike potential is to a great extent of an intuitive character. It is a comparison of these formulas with experimental facts that can determine the degree of confidence to be assigned to the former. This is discussed in the following section.

3. *Experimental Test of the Formula for the Velocity of Conduction in an Axon.*

Indicate with ϕ the diameter of the membrane and putting $r_a = \pi\phi r_M$, the resistance of the membrane in ohm cm², $L_a = \pi\phi L$ its inductance in henry cm² and $c_a = c/\pi\phi$ its capacity in farad/cm², all three per unit area of the membrane. The velocity of conduction can be written as follows:

$$v = \omega \sqrt{\frac{2}{\pi\phi (r_E + r_I) U}}, \tag{9}$$

where $\omega = 2\pi/T$ with T the duration of the spike potential, r_E and r_I the external and internal resistances per unit length, and

$$U = \left[\left(\frac{r_a}{r_a^2 + \omega^2 L_a^2} \right)^2 + \left(\omega c_a - \frac{\omega L_a}{r_a^2 + \omega^2 L_a^2} \right)^2 \right]^{1/2} - \frac{r_a}{r_a^2 + \omega^2 L_a^2}. \tag{10}$$

Until the present time there have been essentially three formulas for the velocity of conduction in an axon; that of N. Rashevsky (1933, 1948), of W. A. H. Rushton (1937) and of F. Offner, A. Weinberg and G. Young (1940).

Experimental data covering mainly non-myelinated fibers indicate that r_a and c_a do not vary greatly for axons of a given kind un-

der constant environment and excitability conditions (Cole and Curtis 1939, 1950; Hodgkin, 1947a; Hodgkin and Rushton, 1946; Cole and Baker, 1941; Cole and Hodgkin, 1939). This is particularly true for membrane capacity which seems to be contained within a narrow range, even for a broad variety of cells (Cole, 1947, p. 23). The spike duration T seems to be constant also for axons of a given kind under constant conditions. The mammalian nerve fibers offer a convincing example of this (Grundfest and Gasser, 1938; Gasser and Grundfest, 1939; Grundfest, 1940; Gasser, 1941; Lloyd, 1947). Thus U seems to be independent of the size of the fiber and for non-myelinated fibers ϕ can be taken as the diameter of the axoplasm and of the external medium at the same time, since the membrane is very thin (see sec. 4 and Cole, 1947, p. 22). Then r_I is inversely proportional to ϕ^2. If r_E is also taken approximately as inversely proportional to ϕ^2, *velocity* appears to be from equation (9) *directly proportional* to $\sqrt{\phi}$. Formulas of Rashevsky and of Offner, Weinberg and Young lead to exactly the same conclusion which has been confirmed by experiments on the squid giant axon. These experiments gave a proportionality of v to $\phi^{0.574}$ (Pumphrey and Young, 1938) with about the same relationship also holding for other Cephalopods.

In myelinated fibers of cat and rabbit nerve the velocity is a linear function of the diameter according to J. B. Hursh (1939) or slightly less than that according to H. S. Gasser and H. Grundfest (1939). However, the previous reasoning which led us to conclude that the velocity of conduction is proportional to the square root of the diameter does not apply here. The conductor AB, through which the current flows transversally (cf. Fig. 1) has been identified in non-myelinated fibers with the cell membrane, but in medullated fibers it includes also the myelin sheath, at least insofar as the resistance of this transversal part of the conductor is concerned (cf. Cole and Hodgkin, 1939; Rosenberg and Schnauder, 1923). But myelin may be as much as 25% of the diameter of the axon, and even more (see, e.g., Kiss and Mihálik, 1928); consequently ϕ cannot be taken at the same time as the diameter of the membrane, of the axoplasm and of the external medium. If these three diameters were proportional to each other, v would be, theoretically, still proportional to $\sqrt{\phi}$; however, experimental data for the fibers of the saphenous nerve of cat and rabbit show that the proportionality between the external and internal diameters holds only for a partial range of these fibers (Gasser and Grundfest, 1939). This non-linearity was

noted also by F. O. Schmitt and R. S. Bear (1937) on fibers of frog sciatic nerve, by Arnell (1936) on spinal nerve and was recently confirmed through extensive studies of G. W. Taylor (1940, 1941, 1942, 1943), F. K. Sanders (1948), Sanders and D. Whitteridge (1946) on nerve fibers of various animals. As Schmitt and Bear state (1939), " . . . comparison of fibre velocity with diameter alone, without regard to degree of myelination, is apt to be confusing." Aside from this it is doubtful whether the structure consisting of membrane and myelin sheath can be represented by the circuit AB of Figure 1 (see sec. 4). Assigning a special function to the Ranvier nodes in the conduction process, Offner, Weinberg and Young (1940) obtained a different argument for a deviation of the conduction velocity from the proportionality relation $v \equiv \sqrt{\phi}$. However, viewpoints on the role that Ranvier nodes may play in the conduction are conflicting (Curtis and Cole, 1940, p. 593; Rosenblueth, Wiener, Pitts and Garcia Ramos, 1948, p. 310; Grundfest, 1947, p. 488), and the assumption of Offner, Weinberg and Young that conduction velocity depends on internodal distance is not confirmed by the experiments of Sanders and Whitteridge (1946).

Together with the formulas of Rashevsky, Rushton and Offner, Weinberg and Young the present formula has in common *the inverse square root proportionality of the velocity to the sum of the external and internal resistances.* That the velocity of conduction should decrease when the external resistance increases was shown by A. L. Hodgkin (1939) on crab and squid giant axon using sea water, air and oil as external media. A quantitative indication of the correctness of this relationship was obtained by B. Katz (1947) using different electrolytic concentrations of the external medium of a crab nerve fiber. An indirect confirmation of the same relation was recently also given by Hodgkin (1947a), and in allied fields of muscle and Nitella cell the qualitative character of the relation has been known for some time (Pond, 1921; Auger, 1933).

The discussion just presented implies the assumption that the spike duration T is independent of the external medium. Although no systematic data seem to be available on this subject, a few records published by Hodgkin (1939) indicate such independence.

The relation (9)—(10) differs from the Offner, Weinberg and Young formula in the way that v is made dependent on the shape of the spike potential. *The present formula makes* v *dependent on the duration of the spike,* whereas Offner, Weinberg and Young make v dependent on its magnitude at the inflection point of the rising phase. This inflection point is quite often unobtainable with suf-

ficient accuracy, since the spike potential sometimes is practically a straight line around that point. As a matter of fact H. S. Gasser and H. Grundfest (1939) calculated the spike potential of a nerve, in good agreement wits its actual shape, by assuming a straight triangular spike for the fibers (cf. also Lloyd, 1947, p. 102). On the contrary, the spike duration is obtainable with quite good accuracy. In addition to this, and still more important, is the fact that the conduction velocity does not seem to be very much affected by variations of spike duration (see numerical results of this section concerning crab axon and an approximate formula for the conduction velocity in Nitella cell).

We proceed now to a numerical computation of the velocity of conduction. Let us consider first a *squid giant axon.* We can take as an average value of the spike duration $T = 2$ msec (cf. Pumphrey and Young, 1938; Hodgkin, 1939; Katz, 1947). K. S. Cole and H. J. Curtis (1940) give for a squid giant axon of 446μ diameter in sea water $r_E + r_I = 37{,}000$ ohm/cm. Let us consider a diameter of 292μ for which values of spike velocity are available (Pumphrey and Young, 1938). We take: $r_E + r_I = 37{,}000 \times (446/292)^2 \approx 86{,}400$ ohm/cm, $L_a = 0.2$ henry cm^2 for the membrane inductance, $c_a = 1\mu$ farad/cm^2 for its capacity, both per unit area (Cole and Baker, 1941; Curtis and Cole, 1944; Cole and Curtis, 1939). To see the effect of inductance and of capacity, calculations are made also with $L_a = 0$ and $c_a = 2\mu$ farad/cm^2. Table I gives the results for various values of the membrane resistance.

TABLE I

CALCULATED CONDUCTION VELOCITIES OF A SQUID GIANT AXON

r_a ohm cm^2 / v m/sec with	25	100	200	400	700
$c_a = 1\ \mu$ f/cm^2, $L_a = 0.2$ h cm^2	12.9	13.5	13.9	13.3	11.9
$c_a = 1\ \mu$ f/cm^2, $L_a = 0$	44.9	22.8	16.6	12.8	11.1
$c_a = 2\ \mu$ f/cm^2, $L_a = 0.2$ h cm^2	7.3	7.5	7.5	7.5	7.2

The experimental value of v given by R. J. Pumphrey and J. Z. Young (1938) for an axon of 292μ is 14.9 m/sec. However, if the smooth curve given by the same authors is used, a magnitude of about 13.5 to 13.6 m/sec is obtained. This is in very good agreement

with the theoretical values of Table I, since the range of 25 to 400 ohm cm^2 is very likely to contain the correct average membrane resistance of the active part of the axon (Cole and Curtis, 1939, 1940; Cole and Hodgkin, 1939; Cole and Baker, 1941).

The results shown in Table I are quite significant. First of all we note that *a change of the membrane resistance* within its range as known through experimental data (Cole et al) *has practically no effect on conduction velocity.* (See the second line of Table I. Average membrane resistances larger than 400 ohm cm^2 are not likely to correspond to axons under action potential.) This may explain why fluctuations in the measured values of r_a (Cole et al; Hodgkin and Rushton, 1946) seem to be higher than those of the conduction velocity. This result is in contrast with that of Offner, Weinberg and Young, which gives v as inversely proportional to $\sqrt{r_a}$. However, r_a has in the present work a somewhat different meaning than in the Offner, Weinberg and Young formula. In the present formulas it is reasonable to consider r_a as an average resistance of the active part of the membrane; in Offner, Weinberg and Young's theory r_a is the local membrane resistance at the moment at which the potential goes through its inflection point in the rising phase of the spike.

Although 1μ farad/cm^2 is a good representative average of membrane capacity, occasionally higher values have also been found. It is seen from Table I that increasing the capacity by a factor of 2 reduces the velocity in the same proportion. This is in agreement with Offner, Weinberg and Young's formula according to which conduction velocity is inversely proportional to capacity. Qualitatively this relationship is intuitive since larger capacity implies a slower charging and discharging process which is believed to accompany the spike conduction.

Another interesting conclusion that can be drawn from Table I is that concerning the *inductance which is of small effect for higher membrane resistances but reduces the conduction velocity in a very pronounced manner at lower membrane resistances. In this way the conduction velocity is maintained practically independent of variations of the membrane resistance.* (Compare lines two and three of Table I). That such a velocity-regulating agent should exist in the membrane is suggested also by the following consideration independent of the concept of nerve inductance: All formulas for the conduction velocity which do not take into account the inductance, including the present one if $L_a = 0$, agree on the fact that this velocity depends on membrane resistance. Now it has been shown by Cole

and Curtis (1939) that membrane resistance changes during activity, lower resistances corresponding to higher degrees of activity (see also Hodgkin and Rushton, 1946, p. 477). Consequently, to each ordinate of the spike potential corresponds a different membrane resistance (see, in particular, Fig. 10 in Cole and Curtis, 1939). Therefore, in the absence of a speed-regulating agent, each ordinate of the spike potential would move at a different speed, which would result in a distortion of the spike potential during conduction. As Table I shows the *inductance slows down high conduction velocities and leaves low velocities practically unaffected.* This may be explained by the fact that inductance stores the energy when its availability increases and releases it later in the decreasing phase. *Through this speed controling function the inductance prevents distortion of the spike potential which would occur during conduction because of a dependence of the conduction velocity on membrane resistance and dependence of the latter on the local magnitude of the potential. Thus inductance would accomplish in a nerve the same function as it does in a telephone cable, although through a different electrical mechanism* (Pupin coils, see, e.g., Knowlton, 1949).

The velocity controling effect of the inductance appears clear from the following expansion of $1/\sqrt{U}$ to which v is proportional:

$$\frac{1}{\sqrt{U}} = \frac{1}{\sqrt{\omega c_a}}\left[1 + \frac{1}{2\omega c_a}\cdot\frac{1}{\omega L_a} + \frac{1}{2\omega c_a}\left(\frac{3}{4}\frac{1}{\omega c_a} + r_a\right)\cdot\frac{1}{(\omega L_a)^2} + \cdots\right] ;$$

This expansion [see eq. (10)] is valid for large inductances. An infinite inductance would make the velocity exactly independent of the membrane resistance (and inversely proportional to the square root of the capacity). A finite, but large, inductance would not change this independence of velocity and membrane resistance by very much, since the latter does not appear in the above expansion below the term of the second order. Even there its effect is not very large, since for the squid giant axon just considered $3/4\omega c_a$ is about 250, and the average membrane resistance during activity is certainly not more than that. As a matter of fact, an infinite inductance would give velocity only about one-third lower than the actual one found with 0.2 henry cm^2. Thus a *possible meaning has been found for the very high inductance which has been noted in the squid giant axon membrane* (Cole et al): *the velocity regulating function of the inductance requires a large amount of the latter.*

The effectiveness of the inductance to keep the conduction velocity independent of variations of membrane resistance appears clear if one considers $L_a = 0$. Then:

$$U = (r_a^{-2} + \omega^2 c_a^2)^{1/2} - r_a^{-1}.$$

Since in the present case ωc_a is about 3×10^{-3} farad/cm² sec and r_a does not exceed 100 ohm cm² by very much, we obtain by power series expansion:

$$U^{-1/2} \approx (2/r_a)^{1/2}/(\omega c_a),$$

which is infinite for $r_a = 0$ and equal to about 47 for $r_a = 100$. A large inductance levels off this variation to a constant value of $1/\sqrt{\omega c_a} \approx 18$.

Of course there is no reason to believe that this may be the only effect of the inductance. It has been noted, for instance, (Eccles, 1948, p. 102) that axons have a decreasing tendency toward oscillatory responses in the face of increasing membrane resistances; this may also be a combined effect of inductance and membrane resistance.

The question of how the relatively high inductance of 0.2 henry cm² can be located in such a small structure as an axon membrane is difficult to answer in a definite manner. Cole and Baker obtained evidence for the existence of an inductance by applying the theory of linear electric circuits. Since ions are the carriers of the current in the nerve, the existence of a high inductance means, within the framework of that theory, the existence of ionic paths which give a high magnetic flux through the membrane. Cole and Baker found that fresh axons never had inductance and that this arose, in a great majority of axons, locally at least, within one hour or so after excision. One could think, therefore, that the conductivity changes, which are known to occur after excision and can take place only through the intermediary of ions, simultaneously bring about the establishment of ionic paths across the membrane which confer to the latter the property of a relatively high inductance. As Cole suggested (1947) changes in conductivity and appearance of the inductance may be closely associated with each other. However, computations of the inductance by standard methods of the theory of electricity (sec. 4) do not indicate the existence of such ionic paths. Consequently, more plausible seems to be the conjecture that the circuit is actually nonlinear and behaves, within a certain range, as if it were linear and had an inductive membrane (Cole, 1941, 1947). As a possible origin of such non-linearity appropriate electrochemical reactions (Bart-

lett, 1948) and piezoelectric effects (Cole, 1941) have been suggested.

TABLE II
CALCULATED CONDUCTION VELOCITIES OF A CRAB AXON

r_a ohm cm²	25	100	1000	7653
v m/sec with				
$T = 1.2$ msec, $L_a = 0$	8.38	4.35	2.46	2.28
$T = 1.2$ msec, $L_a = 0.2$ h cm²	2.47	2.49	2.47	2.29
$T = 2$ msec, $L_a = 0$	8.43	4.25	2.01	1.78

Table II gives the results of calculations on a *crab nerve axon* of 31.8 μ diameter for which data of Hodgkin (1947a) are available; for an axon in oil $r_I/r_E = 1.58$, resistivity of axoplasm = 90 ohm cm, $r_a = 7653$ ohm cm² (axon in resting conditions), $c_a = 1.112$ μ farad/cm². This gives $r_E + r_I = 1.85 \times 10^7$ ohm/cm. From another paper by Hodgkin (1939) we take $T = 1.2$ msec for an axon under similar experimental conditions. It is seen from Tables I and II that the relative effect of an inductance of 0.2 henry cm² would be about the same here as in the squid giant axon. Although the only measured inductances that are available are those of the squid giant axon, Hodgkin and Rushton (1946) noted in some lobster axons a possible inductance of 0.3 henry cm².

The experimental value for conduction velocity in oil of a crab axon of about 30μ diameter, as determined by Hodgkin, is 3.5 m/sec. This suggests, according to Table II, that probably no inductance existed in the membrane and that its average membrane resistance was somewhere between one hundred and a few hundreds of ohm cm². Table II also contains results with a spike duration of 2 msec, which has been observed on crab axon by Hodgkin and Rushton (1946, p. 477). It is seen that a change of T from 1.2 to 2 msec has practically no effect on v, particularly in the pertinent range of r_a.

For *muscle fiber* the data are less complete. Using the values given by B. Katz (1948) for the Extensor Longus Dig. IV of *Rana temporaria*, $\phi = 45\mu$, $c_a = 4.5\mu$ farad/cm², $r_I/r_E = 1.08$, resistivity of fiber interior 260 ohm cm, and taking tentatively $T = 5$ msec, we obtain $v = 0.4$ m/sec for $r_a = 4{,}000$ ohm cm² and $v = 0.7$ m/sec for

$r_a = 100$ ohm cm^2. The figure of 4,000 ohm cm^2 is indicated by Katz for the resistance of a resting membrane. During activity a much lower resistance may be expected. It is seen that the calculated velocity agrees with the measured one within a factor of about 2 (cf. Katz, 1948, p. 527).

We close this section with a calculation of the conduction velocity for Nitella cell. Since the structure of this cell is, from an electrical viewpoint, more complicated than that of an axon (cf. Osterhout, 1943), the diagram of Figure 1 cannot give more than crudely approximated results. The diameter of this cell may be as large as 500μ (Blinks, 1930; Curtis and Cole, 1937) and we base our calculations on a figure of $\phi = 400\mu$. The duration of the action potential may be as large as about 15 sec (see, e.g., Blinks, 1930; Osterhout, 1935, 1936; Hill and Osterhout, 1935; Osterhout and Hill, 1940) and we take tentatively $T = 10$ sec. We will see later that the order of magnitude of the quantities involved is such that the exact value of T is immaterial. A capacity of 1μ farad/cm^2 seems to be as justified here as in the case of an axon (Curtis and Cole, 1937; Cole and Curtis, 1938). We may indicate for a typical resting membrane resistance 250,000 ohm cm^2 (Blinks, 1930; Curtis and Cole, 1937). However, at the height of activity the membrane resistance may decrease by a factor of 200 (Cole and Curtis, 1938); consequently, we take as an average value for the whole active part of the cell $r_a = 10^5$ ohm cm^2. For the sum of the external and internal resistances we assume the round figure of $r_E + r_I = 10^7$ ohm/cm on the basis of data of Blinks (1930). Then $r_a^2\omega^2 c_a^2$ is so small with respect to unity that we can write, since we neglect L_a,

$$U = [(1 + \omega^2 r_a^2 c_a^2)^{1/2} - 1]/r_a \approx \omega^2 r_a c_a^2/2 .$$

Consequently $v \approx 2/[c_a\sqrt{\pi\phi(r_E + r_I)r_a}]$, which is independent of ω. This approximate formula shows the same type of dependence of v on c_a and r_a as Offner, Weinberg and Young's formula. However, according to these authors, their formula is not in agreement with the experimentally observed conduction velocity of *Nitella*. Substituting the previously mentioned numerical constants into the last equation, we obtain $v \approx 5.6$ cm/sec, which falls well within the experimental range of 2 to 20 cm/sec (Osterhout, 1936).

4. *The Electric Characteristics of Axon from a Structural Viewpoint.*

Since electric phenomena in the nerve are of an electrolytic nature, the electric properties of an axon are a consequence of its struc-

ture and of its ionic composition. This fact has been previously used to gain some tentative insight into the membrane (Cole, 1947, 1949). The capacity of a thin cylindrical shell of thickness Δ_c is:

$$c_a \approx K/4\pi\Delta_c\,, \tag{11}$$

where K is the dielectric constant. The theory of diffusion of ions in dilute solutions, together with the law of perfect gases, gives us the following expression for the conductivity of an electrolytic medium (Planck, 1890):

$$\sum_i e_i^2 D_i n_i / kT\,,$$

where n_i is the number of ions per cm^3 of kind i, e_i its charge, D_i its diffusion coefficient which does not need to be assumed independent of concentration, k the Boltzmann constant and T the absolute temperature. From the above we obtain, for membrane resistance per unit area,

$$r_a = kT\Delta / \sum_i e_i^2 D_i n_i\,, \tag{12}$$

where Δ is the thickness of the membrane. If, together with this equation, formula (11) is used for membrane capacity, Δ equals Δ_c on the basis of the electric circuit of Figure 1. However, there are theoretical and experimental reasons to suspect that the circuit in Figure 1 may be, at least in some cases, insufficient to represent the actual situation. Davis and Lorente de Nò (1947), Rushton (1937), Rosenberg (1937), Bogue and Rosenberg (1934) and others considered the diagram of Figure 4, or some of its particular cases, as possible electric equivalents of the membrane. According to the cir-

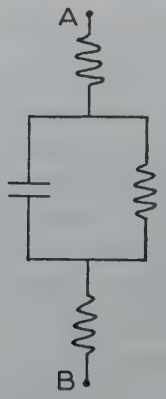

FIGURE 4. Electric circuit illustrating a possible transversal structure in an axon: a heterogeneous membrane or a myelin sheath with an adjacent membrane.

cuit of Figure 4 Δ_c is different from Δ. This differentiation between the two thicknesses may be viewed in the following manner from the standpoint of the theory of diffusion. It is known that one of the prominent characteristics of a living cell membrane is its heterogeneous structure (see, e.g., Davson and Danielli, 1943) through which the membrane acts as a sieve for the diffusing substances. It is conceivable that internally in the membrane, or adjacent to one or both of its surfaces, there is a layer which is impenetrable to some of the ions because of their large size or small energy. These ions would gather on the boundaries of that layer conferring to the latter properties of a capacity; smaller ions or those having a sufficient energy would penetrate the layer, giving rise to a conductance in parallel with the capacity.

Another reason to separately consider a capacity thickness and a conductivity thickness in the transversal structure of the axon arises in myelinated fibers. Electrically, because of their low conductivities, myelin and membrane both behave as transversal structures with respect to the axoplasm, and Δ in formula (12) is the total thickness of this structure. In some of these fibers a high capacity has been observed (Curtis and Cole, 1950, p. 593; Cole and Curtis, 1950, p 88). However, the myelin is so thick that the capacity calculated from formula (11) by putting $\Delta = \Delta_c$ in it would be smaller than the measured one by more than an order of magnitude. Even for a fiber like the squid giant axon, which does not seem to have a myelin sheath in the ordinary sense, but does contain, according to Bear, Schmitt and Young (1937), a similar very thin lipoid structure, calculations would give a capacity that would be much too low if Δ_c in formula (11) were taken as the thickness of this lipoid sheath. In fact, according to these authors, the thickness of this structure is less than 1% of the diameter. Taking, for instance, $\Delta_c = 2.5\mu$, which would be 1/2% for an axon of 500μ, and assuming $K = 3$ (cf. Cole, 1947) we would obtain

$$c_a \approx 3/4\pi \times 2.5 \times 10^{-4} \times 9 \times 10^5\ \mu\text{f/cm}^2 \approx 10^{-3}\ \mu\text{f/cm}^2,$$

which is 1,000 times smaller than the experimental value. This gives support to the diagram of Figure 4 in which one of the two resistances either at A or at B may be zero. Following Cole (1947), formula (11) can be used to determine the thickness of the capacity layer of the membrane. With $c_a = 1\mu\text{f/cm}^2$ the value for Δ_c is 27 angstrom. On the other hand, if the very low capacity of $c = 4.1 \times 10^{-5}\mu$ farad/cm obtained for the peroneal nerve fiber of the bullfrog of

10μ diameter is considered (Davis and Lorente de Nò, 1947) a much thicker capacity layer would be required. From the formula for the capacity of a cylindrical shell we obtain:

$$\Delta_c = (e^{K/2c} - 1)\,\phi/2\,.$$

With $K = 3$ this gives $\Delta_c \approx 2{,}000$ angstrom.

Since, necessarily, $\Delta_c \leq \Delta$, relations (11) and (12) give:

$$\sum e_i^2 D_i n_i \geqslant KkT/4\pi r_a c_a\,. \tag{13}$$

For $K = 3$, $T = 300°$, $c_a = 1\mu$ farad/cm^2, $r_a = 1{,}000$ ohm cm^2, the right-hand side of this inequality is 10^{-11}. Therefore if we call D the average diffusion coefficient and n the total ionic concentration in the membrane, and assume as monovalent the great majority of ions, we obtain $Dn \geqslant 4.3 \times 10^7$ (cm sec)$^{-1}$. If an axon is electrically expressible by the circuit shown in Figure 1, the sign of equality is to be taken in this relation. It is difficult to estimate either one of the two factors on the left-hand side of the above inequality. Cole suggested (1949), on theoretical grounds, $n = 5 \times 10^{17}$ ions/cm^3. This would give approximately $D \geqslant 10^{-10}$ cm^2/sec.

The number of ions which behave statically at the capacity layer can be estimated in the following manner. If E is the voltage across the capacity and Q the charge per unit area, we have $Q = c_a E$. Since $c_a = 1\mu$f/cm^2 we obtain from this, for the number of monovalent ions at each boundary of the capacity layer,

$$10^{-6} \times 3 \times 10^9 \times 10^{-3}\, E/4.8 \times 10^{-10} = 6.25 \times 10^9\, E \text{ ions}\,,$$

where E is now in millivolt. If these ions were arranged regularly in a single layer to form a network with a square-shaped basic unit, the distance a of the two closest neighbors would be:

$$a = 1265/\sqrt{E} \text{ angstrom}\,,$$

which is a very reasonable relationship since for $E = 100$mV the distance would be $a = 126.5$ angstrom.

The product $r_a c_a$ which appears in relation (13) can give us an idea about the rate at which ions flow through the resistance parallel to the capacity, or, phrasing it differently, about the proportion of ions which succeed in penetrating through the capacity layer with respect to those which are too big or too slow to do so. In fact (cf. Cole, 1947)

$$Q = c_a E\,,\; V = r_a I\,,$$

where V is the voltage across the membrane, E that across its ca-

pacity layer, so that $E \leq V$, and I is the intensity of the current flowing across a unit area of the membrane. From the above two equations we obtain:

$$Q/I = c_a r_a E/V \leq c_a r_a .$$

Here again the sign of equality holds if the axon corresponds to the circuit shown in Figure 1. Under this hypothesis *for a squid giant axon* in resting conditions $c_a r_a \approx 10^{-3}$ sec, that is, *per each ion accumulated at the capacity layer there are 1,000 ions per second passing through it. During activity this figure may increase by a factor of 10 or more. Thus the capacity layer seems to be quite easily penetrable by the ions and the fraction of slow or heavy ions seems to be relatively small.* For a Nitella cell c_a is about the same as for the squid giant axon, but r_a is much higher. With $r_a = 10^5$ ohm cm^2 for a resting Nitella cell, ten ions only would flow per second for each one accumulated at the capacity. The discussion presented in this paragraph is based on an idea already considered by Cole (1947) although a misprint makes the conclusion in that paper (p. 51) appear erroneous.

We now proceed to the discussion of the *inductance.* An inductance of 0.2 henry cm^2 for an axon is very high. In fact, the *longitudinal* inductance of a circular cylinder of length l and diameter ϕ is according to Neumann's formula (see, e.g., Rosa and Grover, 1912, p. 150):

$$2l[\ln(4l/\phi) - 0.75] \times 10^{-9} \text{ henry} .$$

For an axon of 400μ diameter and 10 cm length this would give about 1.2×10^{-7} henry, whereas 0.2 henry cm^2 gives a *radial* inductance two million times larger. This comparison suggests that if the 0.2 henry per cm^2 of the membrane are due to a linear conductor the latter would have to give an unusually high magnetic flux. Helically shaped conductors or coils are the most efficient from the inductive viewpoint in the sense that they give high inductance within a small space. Consequently, one could think of only the following way, if any, to materialize through a linear conductor an inductance of 0.2 henry cm^2 within the thin transversal structure of the axon. As it has been pointed out in section 3, changes in conductivity which occur, for instance, after excision of an axon could suggest that new ionic paths are being established in the sheath of the axon. However, even if we assume that these paths are helically shaped, we find that they cannot give the required inductance. In fact, consider the following expression for the inductance of a coil of length Δ, having a

circular cross-sectional area A and N windings per unit length (Rosa and Grover, 1912, p. 116):

$$4\pi N^2 A \Delta \times 10^{-9} \text{ henry}.$$

The inductance per unit area is

$$L = 4\pi N^2 A^2 \Delta \times 10^{-9} \text{ henry cm}^2. \tag{14}$$

Consider a squid giant axon 400μ diameter and let us make assumptions favoring a high L: (i) $N = 2 \times 10^7$ cm^{-1}, that is, a distance of 5 angstrom between two consecutive windings; (ii) $\Delta = 4 \times 10^{-4}$ cm, that is, thickness of the transversal structure of the axon equal to 1% of its diameter, which is possible according to Bear, Schmitt and Young (1937). Then from formula (14) we find that to give 0.2 henry cm^2 the cross-sectional area of the ionic path would have to be $A = 0.01$ cm^2 which is geometrically impossible for an axon of 0.04 cm diameter. In addition to this, even if it were possible it would not correspond to the circuit shown in Figure 1 which assumes a *distributed* current throughout the whole transversal structure and not a guided one in a single circuit of large dimensions.

Thus there is no other choice but to ascribe the apparent inductance to a non-linearity of the circuit.

5. *Kinetics of Electrotonic Phenomena.*

Occasionally the opinion has been expressed that electrotonic phenomena spread instantaneously (cf. Bogue and Rosenberg, 1934). The object of the present section is to examine the speed and the length of spread of the electrotonus, that is, of a subthreshold voltage, in an axon on the basis of its electric characteristics. This analysis is based on the theory recently developed by Hodgkin and Rushton (1946), and by Davis and Lorente de Nò (1947).

If I is the intensity of the current from A to B (Fig. 1), that is, across the membrane, and I_p the intensity of the polarizing current, both per unit length, we have for the *voltage* V *across the membrane due to the current* I_p (Davis and Lorente de Nò, 1947, p. 448):

$$\partial^2 V/\partial x^2 = (r_E + r_I) I - r_E I_p .$$

If c is the capacity and r_M the resistance of the membrane, both per unit length, we have

$$I = J + c\partial V/\partial t ,$$
$$V(x, t) = r_M J(x, t) + L\partial J/\partial t ,$$

where t is the time and J is the intensity of the current through the conductive branch of the circuit from A to B, per unit length (Fig. 1). Putting $J = 0$ for $t = 0$, we obtain from the last equation:

$$J(x, t) = \frac{1}{L} \exp\left(-\frac{r_M}{L} t\right) \int_0^t V(x, \tau) \exp\left(\frac{r_M}{L} \tau\right) d\tau$$

which, substituted in the previous two relations gives an integrodifferential equation for $V(x, t)$. We limit ourselves to the case $L = 0$ and consider a time independent polarizing current. Put:

$$V = \nu I_p r_E \sqrt{\frac{r_M}{r_E + r_I}}, \quad \tau = \frac{t}{c r_M}, \quad \xi = \frac{x}{2} \sqrt{\frac{r_E + r_I}{r_M}},$$

so that ν is a dimensionless measure of the voltage across the membrane, τ and ξ dimensionless measures of time and length. Then we have (Davis and Lorente de Nò, 1947, p. 452):

$$\nu(\xi, \tau) = f(\xi - \xi_a, \tau) - f(\xi, \tau), \tag{15}$$

where $\xi_a < 0$ is the dimensionless abscissa of the anode. Since the cathode is at $\xi = 0$, then

$$4f(y, \tau) = e^{-2|y|} \operatorname{erfc}\left(\frac{|y|}{\sqrt{\tau}} - \sqrt{\tau}\right) - e^{2|y|} \operatorname{erfc}\left(\frac{|y|}{\sqrt{\tau}} + \sqrt{\tau}\right),$$

$$\operatorname{erfc}(u) = \frac{2}{\sqrt{\pi}} \int_u^\infty \exp(-u^2)\, du.$$

Let us consider from now on the *cathodal extrapolar region* $\xi > 0$. The spread $\xi = \xi^*$ of a voltage ν is obtained as the root of the equation

$$2\nu(\xi, \infty) = \exp[-2(\xi - \xi_a)] - \exp(-2\xi). \tag{16}$$

The velocity with which this voltage spreads is:

$$v = \frac{dx}{dt} = \frac{2}{c\sqrt{r_M(r_E + r_I)}} \frac{d\xi}{d\tau},$$

where for each voltage ν, the derivative of $d\xi/d\tau$ is obtained by an implicit differentiation of equation (15). We limit ourselves in these calculations to an *infinite polar distance*; then we have $\xi_a = -\infty$, and $\nu(\xi, \tau) = -f(\xi, \tau)$. From here:

$$\frac{d\tau}{d\xi} = -\frac{\partial f/\partial \xi}{\partial f/\partial \tau} = 2\sqrt{\pi\tau}\left[e^{2\xi}\,\mathrm{erfc}\left(\frac{\xi}{\sqrt{\tau}} + \sqrt{\tau}\right) - 2\nu \right] \exp\left(\tau + \frac{\xi^2}{\tau}\right),$$

where ξ and τ are related to each other by the equation $\nu = \nu(\xi, \tau)$. *At the cathode* $\xi = 0$, and:

$$\frac{d\xi}{d\tau} = \frac{e^{-\tau}}{2\sqrt{\pi\tau}}, \quad \nu = -\tfrac{1}{2}\,\mathrm{erf}\,(\sqrt{\tau}),$$

where $\mathrm{erf}(u) = 1 - \mathrm{erfc}\,(u)$. The elimination of τ between these two equations gives the dimensionless velocity $d\xi/d\tau$ of the dimensionless voltage ν. The result of this elimination is represented in Figure 5. It is seen that low voltages spread with high speed. The

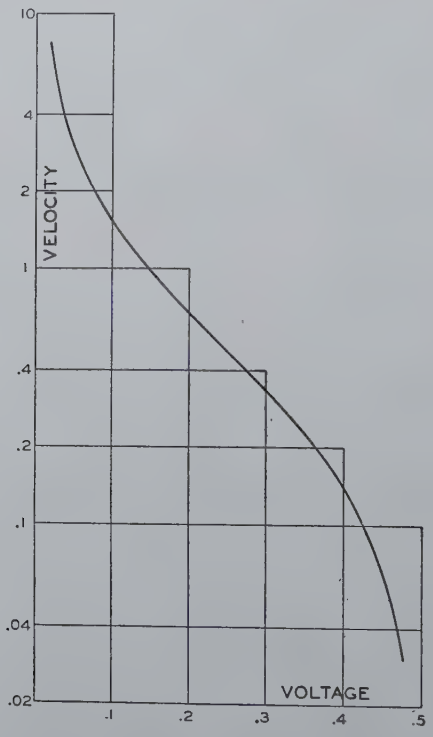

FIGURE 5. Velocity $d\xi/d\tau$ of spread of electrotonic voltage ν at the cathode. Both velocity and voltage in dimensionless units. Infinite polar distance.

extent ξ^* of the spread of the voltage is obtained from equation (16) by putting $\xi_a = -\infty$:

$$\xi^* = -\tfrac{1}{2}\ln(-2\nu). \qquad (17)$$

Since $\nu \leq 0$, voltages which are small numerically have a large spread. $\xi^* = 0$ for $\nu = -1/2$ which is the maximum possible numerical value of ν (cf. Davis and Lorente de Nò, 1947, Fig. 3). Obviously $d\xi/d\tau = 0$ at $\xi = \xi^*$.

As an illustration, let us consider a series of data on squid giant axon (Cole and Hodgkin, 1939): $\phi = 540\mu$, $r_E = 61{,}800$ ohm/cm, $r_I = 16{,}200$ ohm/cm, $r_M = 4{,}110$ ohm cm (which corresponds to about 700 ohm cm^2), $c = 0.17\mu$ farad/cm. Therefore:

$$2/c\sqrt{r_M(r_E + r_I)} = 6.57 \text{ m/sec.}$$

Table III shows some numerical results. For numerically larger ν's, Figure 5 has been used to get $d\xi/d\tau$; for numerically small ν's the asymptotic relations

$$\nu \approx -\sqrt{\frac{\tau}{\pi}}, \quad \tau \approx \pi\nu^2, \quad \frac{d\xi}{d\tau} \approx \frac{\exp(-\pi\nu^2)}{2\pi|\nu|} \approx \frac{1}{2\pi|\nu|}$$

have been employed.

TABLE III
VELOCITY v OF ELECTRONIC
VOLTAGE V AT THE CATHODE

$I_p \mu A$	$-V$ mV	v m/sec
1	0.5	29.8
1	5	1.5
5	0.5	148

Thus it is seen that only very low voltages spread with a high speed. The length of spread of these voltages is respectively 0.6, 0.08 and 1 cm, the largest spread corresponding to the highest velocity v.

SUMMARY

The paper deals with the kinetics of propagation of excitation. It is not concerned with the mechanism through which the action potential arises. The basic idea of the paper is that the velocity of propagation of a perturbation is determined by the electric characteristics of the conductor and by some characteristics of the perturbation. It appears that as far as the spike potential is concerned, its duration, together with the electric properties of the axon, are sufficient to determine the velocity of conduction. The formula derived is in accord with the known facts on the dependence of this velocity

on various parameters. Computed conduction velocities for the squid giant axon, crab nerve axon and Nitella cell agree with experimental measurements. The theory indicates as a possible role of inductance that of slowing down high conduction velocities which would arise in the case of a decrease of membrane resistance. The membrane capacity and conductance are examined from the viewpoint of the theory of diffusion. A quantitative discussion of the inductance confirms the opinion expressed by other authors that inductance is probably an effect of some non-linear electric characteristic of the axon. The velocity of spread of the electrotonic voltage is also analyzed.

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THEORETICAL CONSIDERATION OF A POSSIBLE MECHANISM IN THE CONDUCTION PROCESS OF THIN-SHEATHED NERVE FIBERS

WILLIAM J. FRY AND RUTH BAUMANN FRY
UNIVERSITY OF ILLINOIS
URBANA, ILLINOIS

The propagation of a transverse disturbance along a tubular membrane enclosing a fluid medium and embedded in another is considered. It is shown that the velocity of propagation of such a disturbance can be identified with the velocity of the conduction process of thin-sheathed nerve fibers. The required values of the associated parameters, tension and pressure, appear not unreasonable. The results obtained indicate that experimental observations on the relation between the conduction velocity and the fiber diameter, as well as the effects of longitudinal stretching and transverse squeezing on the velocity of the conduction process in nerve, may be correlated on such a basis.

We consider the possibility of mechanical motion as a factor in the determination of the velocity of the conduction process in thin-sheathed nerve fibers. As indicated in the literature (Hill and Keynes, 1949; Tobias and Solomon, 1950; Solomon and Tobias, 1950; Tobias, 1950), there exists evidence to support the view that mechanical motion is a concomitant of electrical activity in nerve tissue. A mutual coupling between electrical and mechanical changes can be realized, for example, through a piezoelectric or electrostrictive mechanism.

It will be demonstrated in the succeeding analysis that the velocity of propagation of a transverse disturbance along a cylindrical membrane might be identified with the velocity of propagation of the conduction spike of a thin-sheathed nerve fiber. Numerical values of the requisite parameters are obtained during the course of the analysis. The picture developed is also in accord with other aspects of the conduction process. However, since associated electrical phenomena have not been incorporated into the theory the results should be regarded as suggestive in nature at the present time.

In order to develop a picture in which mechanical disturbances play a major role in the determination of the characteristics of the

conduction process of nerve tissue, it appears necessary to consider disturbances of a type which can be localized or guided along a cylindrical membrane. It does not appear feasible to develop a theory on the basis of longitudinal acoustic waves. Such waves would not be guided either along or inside a tube if the values of the acoustic parameters characterizing the external fluid are close to those of the internal fluid, when the tube has the properties of a thin membrane. Furthermore, the velocity of propagation of longitudinal waves in a liquid medium such as water is about 1500 meters per second. This is of the order of 50 times greater than any observed conduction velocities in invertebrate nerve fibers. Since the density of the material present in the nerve is in the neighborhood of 1 gram/cm^3 the compressibility of the medium would have to be very much different from that of water solutions. Assuming that one could surmount these limitations, the variation of the conduction velocity with the radius of the fiber would lead to further complication.

It is possible to free one's self from all the above difficulties by developing a picture on the basis of the transverse vibrations of a

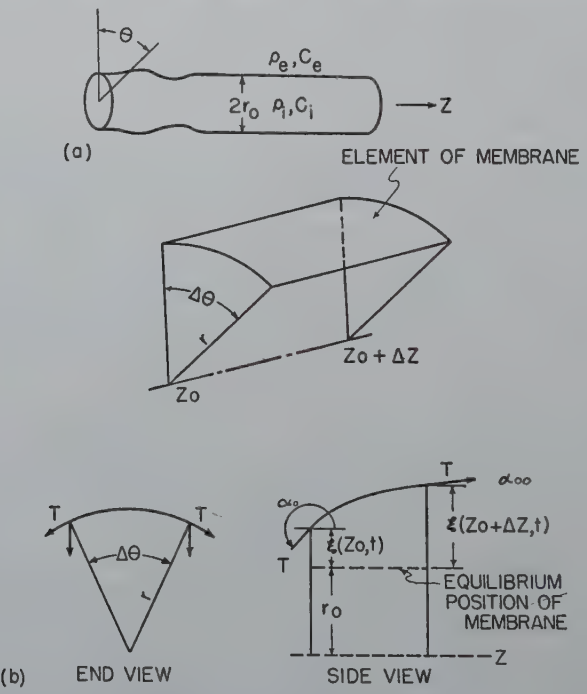

FIGURE 1. (a) A transverse disturbance on a cylindrical membrane of circular cross section. (b) Details indicating forces on an element of the membrane.

tubular membrane enclosing a liquid and embedded in an external fluid medium. A possible role of shear waves in the nerve conduction process has been considered by W. Sutherland (1905, 1906–07, 1908–09). The investigations of O. Frank (1926) on the vibrations of the walls of blood vessels are of interest in this connection, but are not sufficiently general to include the analysis presented here.

We consider a cylindrical membrane of circular cross section as illustrated in Figure 1a. The membrane is under tension, of magnitude T per unit length, perhaps as the result of an excess internal pressure. Its mass per unit area is designated by σ.† The density and acoustic velocity of the internal fluid are represented by ρ_i and c_i, respectively. The symbols ρ_e and c_e designate the corresponding quantities for the external fluid. We are concerned with the equations appropriate for discussing transverse vibrations of small amplitude on such a membrane with the associated disturbances in the surrounding media. It is sufficiently general for our present purpose to restrict the detailed discussion to a symmetrical disturbance, that is, one independent of the angle θ (Fig. 1).

Let us consider a small section of such a membrane as illustrated in Figure 1b. Let ξ represent the displacement of the membrane in the radial direction and let ΔP be the difference between the internal and external pressures, i.e., $\Delta P = P'_{i0} - P'_{e0}$. Then the forces acting on this element in the radial direction are

(i) $$T r_{z_0} \Delta\theta \sin\alpha_0 + T r_{z_0+\Delta z_0} \Delta\theta \sin\alpha_{00} \simeq T \Delta\theta r_0 \left(\frac{\partial^2 \xi}{\partial z^2} \right)_{z_0} \Delta z$$

over the two arcs at z_0 and $z_0 + \Delta z_0$;

(ii) $$-T \Delta z \Delta\theta$$

over the two elements parallel to the z axis, and

(iii) $$\Delta P r_0 \Delta\theta \Delta z = (P'_{i0} - P'_{e0}) r_0 \Delta\theta \Delta z$$

because of the difference in pressure across the membrane. The dynamical equation then follows:

$$\frac{\partial^2 \xi}{\partial z^2} - \frac{1}{r_0} + \frac{P'_{i0} + P'_{e0}}{T} = \frac{\sigma}{T} \frac{\partial^2 \xi}{\partial t^2}. \tag{1}$$

Now the difference $P'_{i0} - P'_{e0}$ can be written as

$$P'_{i0} - P'_{e0} = \Delta P_0 + P_{i0} - P_{e0}, \tag{2}$$

†For list of symbols and their definitions see page 314.

where P_{i0} and P_{e0} are the deviations in pressure from the equilibrium values in the two media at the surface of the membrane caused by the disturbance on the membrane, and ΔP_0 is the pressure difference at equilibrium. The relation between the tension T and the equilibrium pressure difference ΔP_0 is given by

$$T = r_0 \Delta P_0 . \tag{3}$$

Expression (1) then reduces to

$$\frac{\partial^2 \xi}{\partial z^2} + \frac{P_{i0} - P_{e0}}{T} = \frac{\sigma}{T} \frac{\partial^2 \xi}{\partial t^2}. \tag{4}$$

The same differential equation (4) is obtained if the tension is maintained by forces other than those arising from a hydrostatic pressure difference. Under the assumption that the displacement of the membrane is small, the equation appropriate for discussing the disturbance in the fluid media is the usual acoustic wave equation. We tabulate it here expressed in the symbols for the internal and external fluids:

$$\begin{aligned} \nabla^2 P_i &= \frac{1}{c_i^2} \frac{\partial^2 P_i}{\partial t^2}; \\ \nabla^2 P_e &= \frac{1}{c_e^2} \frac{\partial^2 P_e}{\partial t^2}, \end{aligned} \tag{5}$$

where P_i and P_e are the pressure deviations from the equilibrium values at any point in the media. The effect of viscosity is neglected. Usually the inclusion of viscous terms affects the expression for velocity only slightly and introduces an amplitude damping factor. An indication of the magnitude of the damping, resulting from viscosity, for small amplitude disturbances of the type discussed might be given by the damping factor of an acoustic disturbance of the same frequency. On the basis of observations at ultrasonic frequencies, it appears that the damping factor for the frequencies and path lengths involved here would be small. Since the membrane is circular in cross section, we express the Laplacian operator in cylindrical coordinates. (See, for example, Morse, p. 296.) It is convenient first to discuss solutions to (4) and (5) which are sinusoidal functions of time. We write

$$\begin{aligned} \xi &= \xi_A e^{j\omega t}; \\ P_i &= P_{iA} e^{j\omega t}; \\ P_e &= P_{eA} e^{j\omega t}. \end{aligned} \tag{6}$$

The expressions (4) and (5) then become

$$\frac{d^2\xi_A}{dz^2}+\omega^2\frac{\sigma}{T}\xi_A=-\frac{P_{iA0}-P_{eA0}}{T};$$

$$\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial P_{iA}}{\partial r}\right)+\frac{\partial^2 P_{iA}}{\partial z^2}+\left(\frac{\omega}{c_i}\right)^2 P_{iA}=0; \qquad (7)$$

$$\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial P_{eA}}{\partial r}\right)+\frac{\partial^2 P_{eA}}{\partial z^2}+\left(\frac{\omega}{c_e}\right)^2 P_{eA}=0.$$

A solution to the equations (7) which satisfies the requirements that (a) it approach zero as r approaches infinity and (b) there be no singularity at $r=0$ is the following:

$$\begin{aligned}\xi_A&=a\,e^{-jk_z z},\\ P_{iA}&=A_i\,I_0[(k_z^2-(\omega/c_i)^2)^{1/2}r]\,e^{-jk_z z},\\ P_{eA}&=A_e\,K_0[(k_z^2-(\omega/c_e)^2)^{1/2}r]\,e^{-jk_z z},\end{aligned} \qquad (8)$$

where a, A_i and A_e are constants to be related to one another through the boundary conditions; I_0 and K_0 are Bessel functions of zero order which are especially suited to the problem since $k_z > \omega/c_i$ or ω/c_e. This latter relation is obtained as follows, $k_z=\omega/V_\omega$ where V_ω is the phase velocity for a sinusoidal disturbance traveling along the membrane. We are interested in phase velocities which are, in general, much smaller than the velocity of sound in the fluid media. This determines the direction of the above inequality.

In order to interrelate the amplitude constants and to determine the propagation constant k_z, we must apply the appropriate boundary conditions at the surface of the membrane, that is, the radial velocities of the fluid media at $r=r_0$ must equal the membrane velocity. Now the relation between the radial velocity, u_r, and the pressure is (see, for example, Morse, 1948, chap. v; note the sign change due to the choice of $e^{-j\omega t}$ in place of $e^{j\omega t}$ of this paper):

$$u_r=\frac{j}{\omega\rho}\frac{\partial P}{\partial r}. \qquad (9)$$

We obtain, therefore, the expression

$$\frac{j}{\omega\rho_i}A_i\left(\frac{dI_0}{dr}\right)_{r_0}e^{-jk_z z}=j\omega a e^{-jk_z z}=\frac{j}{\omega\rho_e}A_e\left(\frac{dK_0}{dr}\right)_{r_0}e^{-jk_z z}. \qquad (10)$$

Consequently,

$$A_i = \frac{\omega^2 \rho_i a}{\left(\frac{d I_0}{d r}\right)_{r_0}} \quad \text{and} \quad A_e = \frac{\omega^2 \rho_e a}{\left(\frac{d K_0}{d r}\right)_{r_0}}. \tag{11}$$

But (see, for example, Watson, 1944, p. 79)

$$\left(\frac{d I_0}{d r}\right)_{r_0} = (k_z^2 - (\omega/c_i)^2)^{1/2} (I_1)_{r_0}$$

$$\text{and} \left(\frac{d K_0}{d r}\right)_{r_0} = - (k_z^2 - (\omega/c_e)^2)^{1/2} (K_1)_{r_0}.$$

Therefore,

$$P_{iA0} = \frac{\omega^2 \rho_i}{(k_z^2 - (\omega/c_i)^2)^{1/2}} \left(\frac{I_0}{I_1}\right)_{r_0} \xi_A$$

and (12)

$$P_{eA0} = \frac{-\omega^2 \rho_e}{(k_z^2 - (\omega/c_e)^2)^{1/2}} \left(\frac{K_0}{K_1}\right)_{r_0} \xi_A.$$

Upon substituting (12) into the first of equations (7), we obtain

$$\frac{d^2 \xi_A}{d z^2} + \omega^2 \left\{ \frac{\sigma}{T} + \frac{\rho_i}{T (k_z^2 - (\omega/c_i)^2)^{1/2}} \left(\frac{I_0}{I_1}\right)_{r_0} + \frac{\rho_e}{T (k_z^2 - (\omega/c_e)^2)^{1/2}} \left(\frac{K_0}{K_1}\right)_{r_0} \right\} \xi_A = 0. \tag{13}$$

Since the first equation of (8) is a solution of (13), we have the following implicit expression for k_z

$$k_z^2 = \omega^2 \left\{ \frac{\sigma}{T} + \frac{\rho_i}{T (k_z^2 - (\omega/c_i)^2)^{1/2}} \left(\frac{I_0}{I_1}\right)_{r_0} + \frac{\rho_e}{T (k_z^2 - (\omega/c_e)^2)^{1/2}} \left(\frac{K_0}{K_1}\right)_{r_0} \right\}. \tag{14}$$

The phase velocity V_ω can then be expressed implicitly as

$$V_\omega^2 = 1 \Big/ \left\{ \frac{\sigma}{T} + \frac{\rho_i}{T(k_z^2 - (\omega/c_i)^2)^{1/2}} \left(\frac{I_0}{I_1} \right)_{r_0} + \frac{\rho_e}{T(k_z^2 - (\omega/c_e)^2)^{1/2}} \left(\frac{K_0}{K_1} \right)_{r_0} \right\}. \tag{15}$$

As indicated above, we are primarily concerned with phase velocities which are much less than the velocities c_i and c_e. In addition, since $\sigma << \rho_i$, we can neglect the first term by comparison with the other two. For such situations, we can write (15) as

$$V_\omega^3 = \omega \frac{T}{[\rho_i (I_0/I_1)_{r_0} + \rho_e (K_0/K_1)_{r_0}]}, \tag{16}$$

where the argument of the Bessel functions is $(\omega r_0/V_\omega)$. Now the magnitude of this argument increases with increasing values of both r_0 and ω. Let us consider, then, relatively large values as follows: $r_0 = 2(10)^{-2}$ cm, $V_\omega = 2(10)^3$ cm/sec and $\omega = (10)^5$ sec^{-1}. The first value is the approximate radius of a giant squid axon, the second is the conduction velocity in the axon and the third value corresponds to a frequency component $\sim 2(10)^4$. From (16) we then obtain the result that for equal densities of the interior and exterior fluids the value of the second term in the denominator is only about one-third the value of the first term. If the exterior fluid is then replaced by a fluid of very low density (gas) the phase velocity will change by less than 10%. For a value of $\omega = (10)^4$, the phase velocity will change by about 2% for the same exchange. We can thus conclude that the density of the external fluid is of minor importance in determining the velocity of propagation.

If we restrict the range of ω to values such that $\omega \leq (10)^5$, then we can rearrange expression (16) in the following approximate form

$$V_\omega^4 = \frac{\omega^2 T r_0}{2 \rho_i}. \tag{17}$$

We see that the phase velocity is proportional to the square root of ω, i.e., the membrane behaves like an anomalously dispersive medium. In other words, the group velocity of a wave packet is greater than the phase velocity of the midfrequency component. In this case $V_g = 2V_0$. If we substitute from expression (3) for T, we obtain the relation

$$V_\omega^4 = \frac{\omega^2 r_0^2 \Delta P_0}{2 \rho_i}. \tag{18}$$

If the tension is the result of a mechanism other than a hydrostatic pressure difference it appears necessary to postulate $T \propto r_0$. The quantity ΔP_0 in (18) is then replaced by a proportionality constant. Now if we assume that the quantity ΔP_0 is constant, i.e., independent of r_0, the result for the phase velocity is

$$V_\omega = r_0^{1/2}\, \omega^{1/2} \left(\frac{\Delta P_0}{2\, \rho_i} \right)^{1/4}. \tag{19}$$

The third equation of (8) indicates that the rapidity with which a disturbance drops off as one moves radially away from the surface of the tube is greater for the larger values of ω. For example, if one inserts the values $r_0 = 2(10)^{-2}$ cm, $V_\omega = 2(10)^3$ cm/sec and $\omega = (10)^5$ sec^{-1}, one computes from the last equation of (8) that at one diameter distance from the membrane the amplitude of the disturbance is only 1/10 the amplitude at the membrane.

For purposes of comparison with experiment, we are, of course, interested in the velocity of propagation of a fairly localized disturbance along the length of the tube. We will show that as long as a signal velocity has a meaning for any particular disturbance, this velocity depends on the radius r_0 in the same way that the phase velocity depends on r_0. This is shown with the use of the Fourier transform. (See, for example, Guillemin, 1935, chap ii.) We choose an exciting pulse $f(t)$ for the displacement, ξ, of the membrane at $z = 0$. Then this pulse can be represented in terms of sinusoidal functions of time by means of the integral relation

$$f(t_0) = \int_{-\infty}^{+\infty} g(\omega)\, e^{j\omega t}\, d\omega,$$

where (20)

$$g(\omega) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} f(t)\, e^{-j\omega t}\, dt.$$

From the first of equations (8) we see that the disturbance as a function of time will have the following form at a point z_0 to the right of zero (an identical relation in time is observed at z_0 units to the left of zero)

$$f(t)_{z_0} = \int_{-\infty}^{+\infty} g(\omega)\, e^{j\omega(t - z_0/\beta\omega^{1/2})}\, d\omega, \tag{21}$$

where $\beta\omega^{1/2}$ has been inserted for V_ω. The quantity β is a constant. We now refer to equations (17), (18) or (19). If we consider the movement of some point of the disturbance, such as a maximum or

peak value (if this is possible), and define a signal velocity with respect to this point, then, from (21), such a signal velocity, V_s, must satisfy the following relation: $V_S \propto \beta$. We can thus write

$$V_S = b r_0^{1/2} \left(\frac{\Delta P_0}{2 \rho_i} \right)^{1/4}, \tag{22}$$

where b is a constant which is to be determined by evaluation of the integral (21). The relation indicated by (22) between the radius r_0 and the velocity V_S is that observed experimentally.

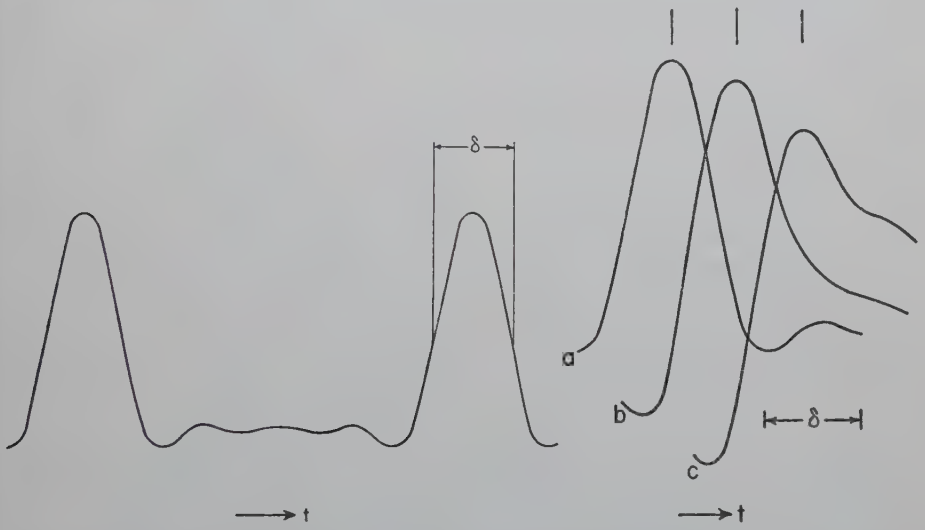

FIGURE 2. (a) Periodic excitation function applied at $z = 0$. (b) The propagated response as a function of position along the tube. The curve labeled a is the exciting function, curve c indicates the response at a point twice as far from the position of excitation as that indicated by curve b.

We have not carried out an exact evaluation of (21) for any pulse shape. However, to obtain a value for the order of magnitude of the signal velocity, we carry out an approximate analysis for a particular exciting function. We assume that the exciting function is a periodic one compounded from four frequencies (multiples of $1/5\delta$) of the form indicated in Figure 2a. Then we obtain for $f(t)$ at the two points to the right of the origin $z_0 = c(\pi\delta/2)^{1/2}$ and $z_{00} = 2c(\pi\delta/2)^{1/2}$, where c is related to the phase velocity of a component corresponding to $\omega = (2\pi/\delta)$ (0.8) by $V_\omega = c[(2\pi/\delta)(0.8)]^{1/2}$, the disturbances labeled (b) and (c) in Figure 2b. It is evident that one can identify a signal velocity if one does not consider points too

far removed from the point of excitation. In this particular case, the signal velocity is equal to about 0.9 of the phase velocity of the highest frequency component present in the exciting function. A calculation based on a value of this order, for a case corresponding to a giant squid axon, yields a value of the required pressure difference, ΔP_0, of the order of several atmospheres. Pressures of this order of magnitude appear somewhat high. However, from formula (18) we see that a doubling of the value of ω decreases the value of the required pressure by a factor of four. The range of values of ω is suggested by the shape of the spike potential and the membrane conductance change with time (Cole and Curtis, 1939).

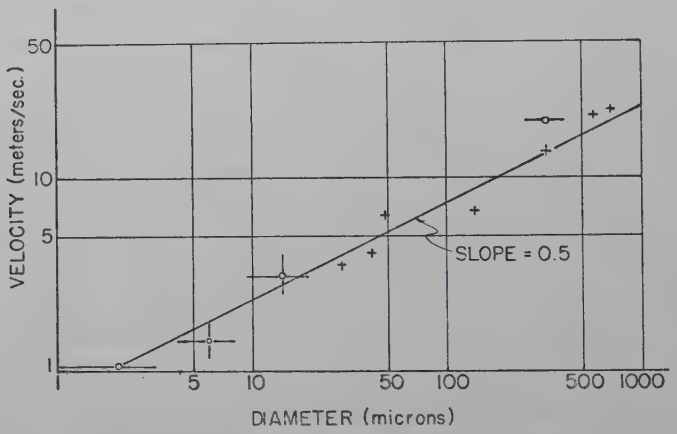

FIGURE 3. Comparison of experimental values for the velocity of conduction versus fiber diameter with the theoretical result.

The graph of Figure 3 indicates a comparison of theoretical results with experiment. The experimental values are from the data tabulated by C. L. Prosser (1946) for crustacea and cephalopod mollusks. The straight line is drawn for a 0.5 power relationship, i.e., the quantity ΔP_0 in (22) is taken constant.

Recent experimental work lends support to the view that mechanical motion coexists with electrical changes taking place in nerve during the conduction process. Dimensional and opacity changes in nerve in the region of electrodes supplying polarizing current have been observed by J. M. Tobias and S. Solomon (1950); Solomon and Tobias (1950) and Tobias (1950). Their results considered with the work of D. K. Hill and R. D. Keynes (1949) on opacity changes accompanying propagated electrical activity suggest the possibility of mechanical motion during activity. The ease of stimulation of nerve tissue by mechanical means also lends support to such a picture

(Blair, 1936).

Investigators (Curtis and Cole, 1942; Hodgkin and Huxley, 1945; Hodgkin and Rushton, 1946) have found useful the concept of an inductive element in the equivalent electrical circuit of nerve. The magnitude of the required inductance is large, of the order of 0.2 H cm^2. It is not necessary that an inductive element as such exist in nerve tissue. Indeed, one of such magnitude would be difficult to accept; mechanical motion, however, would manifest itself in the equivalent electrical circuit of the tissue as a relatively large inductive element.

It is an immediate consequence of (17) and (20) that as the tension varies so does the signal velocity. Experimental results indicate that the conduction velocity may increase when a nerve is longitudinally stretched (Bullock, 1945). Such stretching would increase the tension, T, in relation (17) and thus increase the velocity if the change in radius r_0 does not over-compensate. It would be revealing to check the theory against precise quantitative data on this effect. The experimental evidence that lateral squeezing reduces the conduction velocity (Gasser and Erlanger, 1929) is a direct consequence of (17) since the tension, T, would be reduced in the region of lateral compression. This theory is not inconsistent with the results presented by H. Grundfest (1936) on the effects of hydrostatic pressure. No change in the conduction velocity would be observed on this picture if the difference between internal and external pressures remains constant.

The theory presented in this publication is entirely mechanical. In order to correlate the electrical manifestations of the conduction process of invertebrate nerve with such a picture, it is necessary to postulate an electromechanical coupling mechanism. The mechanical motion might then serve as a sort of trigger for electrical "breakdown." In any event, such considerations await further investigation.

It is concluded on the basis of the theory developed herein that mechanical motion may be a primary factor in determining the velocity of propagation of the conduction pulse of thin sheathed nerve fibers.

We wish to thank Dr. V. J. Wulff for his interest and for helpful discussions.

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LIST OF SYMBOLS

a, A_i, A_e	amplitude constants
c_i, c_e	acoustic velocity of internal and external fluid respectively
k_z	$= \omega / V_\omega$
ΔP	$= P'_{i0} - P'_{e0}$
P_i, P_e	pressure deviations from equilibrium in the respective media
P_{i0}, P_{e0}	deviations in pressure from equilibrium value at surface of membrane in respective media
P'_{i0}, P'_{e0}	pressure at membrane in the respective media
$P_{iA} P_{eA}$	pressure displacement amplitudes in respective media
P_{iA0}, P_{eA0}	pressure displacement amplitudes in respective media at the membrane surface
r	radial coordinate
r_0	radius of cylindrical membrane
T	tension per unit length in the membrane
t	time
u_r	radial velocity
V_g	group velocity
V_S	signal velocity
V_ω	phase velocity for sinusoidal disturbance on the membrane
z	coordinate, distance along membrane
I_0, K_0, I_1, K_1	Bessel functions order zero and one, respectively
ω	$2\pi f$ where f is the frequency
δ	width of exciting pulse at half amplitude
θ	angular coordinate
ξ	radial displacement of membrane
ξ_A	radial displacement amplitude of membrane
ρ_i, ρ_e	densities of internal and external media, respectively
σ	mass of membrane per unit area

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CONTRIBUTION TO THE PROBABILISTIC THEORY OF NEURAL NETS: III. SPECIFIC INHIBITION

ANATOL RAPOPORT
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

The input-output formula is derived for a neuron upon which converge the axones of two other neurons (one excitatory, the other inhibitory) which are themselves subjected to a "Poisson shower" of excitatory stimuli. If the period of latent inhibition, σ, does not exceed one half the refractory period, δ, the input-output curve has no maximum. If, however, $\sigma > \delta/2$, a maximum exists in the input-output curve. As the outside frequency x increases without bound, the output frequency x_3 approaches an asymptotic value which ranges from $1/\delta$ to 0, depending on the ratio σ/δ. The maximum output (if it exists) is also derived as a function of σ and δ.

In previous papers, I and II of this series (Rapoport, 1950a,b), we derived input-output functions for a single neuron receiving a shower of completely randomized (Poisson-distributed) stimuli. In most examples treated in those papers the stimuli were supposed to come from outside the organism. However, in the case of inhibition, where some of the stimuli were supposed to be excitatory and some inhibitory, it seemed too artificial to suppose that the shower of outside stimuli could be subdivided into two "sub-showers," one excitatory, one inhibitory. We supposed, therefore, that a homogeneous shower of stimuli impinged upon two neurons, and that it was these neurons which produced respectively an excitatory and an inhibitory effect upon a third neuron. The output of the third neuron as a function of the outside input was then derived.

This model was interesting chiefly because of the maximum which characterized the output curve of the third neuron with respect to the outside input. It was shown in I that the existence of such maxima allowed the construction of "filter nets" in which certain neurons would respond only to certain *ranges* of the outside input frequency. Moreover, these ranges could be narrowed to any desired degree by simply raising the thresholds of the neurons. Thus a counterpart of a resonance phenomenon could be accounted for in a neural net.

The weakness of the filter net model discussed in I lay in the drastic simplification of neglecting the refractory period. This allowed the problem to be treated by quite simple mathematical means. In the present paper, it will be supposed that neurons have a finite, constant refractory period δ. The inclusion of a refractory period complicates the mathematical treatment considerably, as will be seen, since the time distribution of firings of the neurons is no longer a Poisson distribution. Moreover, the existence of a maximum in the output (and hence the possibility of using the model for constructing "filter nets") will now depend on a certain relation between the refractory period and the period of latent inhibition.

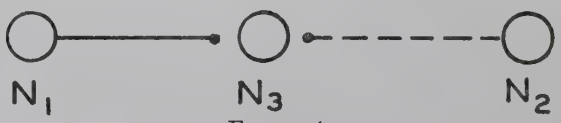

FIGURE 1

Consider a net such as is shown in Figure 1. The firing conditions for such a net in the McCulloch-Pitts notation (McCulloch and Pitts, 1943) would be, under the assumption of quantized time, where the synaptic delay is taken as the unit of time,

$$N_3(t) \cdot\equiv\cdot N_1(t-1) \cdot \overline{N_2(t-1)}. \tag{1}$$

We shall retain the synaptic delay (assumed constant for all neurons) as the unit of time. However, we will not demand that firings occur only at moments which are all multiples of the synaptic delay measured from some common origin, as is supposed in the above-mentioned paper of McCulloch and Pitts. Now if δ designates the refractory period, and σ the period of latent inhibition, we may rewrite equation (1) as follows:

$$\begin{aligned} N_3(t) \cdot\equiv\cdot N_1(t-1) \cdot [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1)}] \\ \cdot [\tau_2 < \sigma \cdot\equiv\cdot \overline{N_2(t-\tau_2-1)}] . \end{aligned} \tag{2}$$

That is to say, "N_3 fires at time t, provided N_1 has fired at time $(t-1)$, and provided N_3 has not fired within the interval δ before t, and provided N_2 has not fired within the period of latent inhibition prior to the arrival of the impulse at N_3 from N_1."

We are interested in the output of N_3 as a function of the average frequency x of the outside stimuli. Evidently, the output will not be the same as that derived in I, where the refractory period was neglected. In that case, the stimuli received by N_3 from N_2 became

infinitely frequent with increasing x, so that almost all of the impulses from N_1 were "canceled" by stimuli from N_2. The limiting output x_3 of N_3 as x increased without bound was zero. As we shall see, this will not be the case with the present model.

Suppose x increases without bound, so that both N_1 and N_2 fire with their limiting frequency $1/\delta$. Since N_1 and N_2 are independent of each other, there is no way of knowing how their firing patterns will "lock" in the limiting frequencies. If they lock so that the firing of N_1 follows that of N_2 at less than σ units, all of the N_1 stimuli will be canceled by N_2, and N_3 will not respond at all. On the other hand if N_1 follows N_2 at more than σ units, N_2 will have no effect, and N_3 will respond *every* time, namely, with the frequency $1/\delta$.

Thus a definite input-output curve for N_3 cannot be derived on this basis. If, however, instead of a single neuron N_2 we consider a whole aggregate of such neurons, then the average output intensity (frequency of firing per neuron) can be computed.† This problem will be considered here.

Firing Distributions of N_1 *and* N_2.

We shall introduce a certain distribution function, which is a generalization of an analogous function in the case of the Poisson distribution. For a Poisson distribution of a sequence of events, the function

$$P(t) = xe^{-xt} \tag{3}$$

represents the probability distribution with respect to the time of occurrence of the next event, where the origin of the time coordinate can be chosen *arbitrarily*. Evidently this distribution does not hold if the "events" are the firings of a neuron with a finite refractory period, since the choice of origin does make a difference in this case. If, for example, the origin is chosen exactly δ units after a firing instant, then, since the neuron is now responsive to every incidence of outside stimulation, the probability distribution of the time of the next firing is indeed given by (3). But if the origin is chosen at a firing instant, then, obviously $P(t) = 0$ for $t < \delta$, so that (3) does not hold.

†Strictly speaking the same reasoning can be applied to a single neuron if the frequency is averaged over sufficiently long periods of time, since there are bound to occur "shifts in phase" in the firing patterns due to random fluctuations in the instantaneous frequency of the outside shower. Such long interval averages seem, however, to be of slight physiological interest, if our definition of the firing frequency of a region involves a more or less instantaneous frequency (short interval average).

We wish to compute the probability distribution of the time of the next firing for N_1 for the case where the choice of origin is *unknown*. For this purpose we introduce a function which represents the probability distribution of the time of the next firing, where it is supposed that the last firing occurred at $t = -y$. This function is given by

$$\begin{aligned} &p(y, t) = 0 \quad \text{for} \quad 0 \leqq t \leqq \delta - y; y < \delta, \\ &p(y, t) = x \operatorname{Exp}\{-x(t - \delta + y)\} \quad \text{for} \quad t > \delta - y; y < \delta, \\ &p(y, t) = xe^{-xt} \quad \text{for} \quad y \geqq \delta. \end{aligned} \tag{4}$$

The function we seek is $P(t)$, the "expected" form of $p(y, t)$, i.e., its "average" with respect to all possible choices of y. Since the probability of the time of occurrence of the "next" event is the same whether time is measured forward or backward, if the choice of origin is not known, it follows that if we choose an instant at random, the probability of choosing an instant between y and $y + dy$ following a firing is $P(y)dy$. Hence to get $P(t)$ we must multiply $p(y, t)$ by the probability of y, $P(y)dy$, and integrate over the entire range of y. This leads to the following integral equation for $P(t)$.

$$P(t) = \int_{\delta - t}^{\delta} P(y) x \operatorname{Exp}\Big\{-x(t - \delta + y)\Big\} dy + \int_{\delta}^{\infty} P(y) xe^{-xt} dy \tag{5}$$

for $t \leqq \delta$;

$$P(t) = \int_{0}^{\delta} P(y) x \operatorname{Exp}\Big\{-x(t - \delta + y)\Big\} dy + \int_{\delta}^{\infty} P(y) e^{-xt} dy \tag{6}$$

for $t > \delta$.

At $t = \delta$ the two expressions are identical, so that $P(t)$ is a continuous function of t everywhere. Equation (6) can be immediately reduced to a differential equation by differentiating both sides with respect to t. In fact,

$$P'(t) = -xP(t), \quad \text{for} \quad t > \delta, \tag{7}$$

which upon integration yields

$$P(t) = Ae^{-xt}, \tag{8}$$

where A is independent of t (is a constant of integration to be computed below).

For $t \leqq \delta$, the situation is somewhat more involved. Differentiation of (5) with respect to t gives a differential-difference equa-

tion,

$$P'(t) = -xP(t) + xP(\delta - t), \quad \text{for} \quad t \leqq \delta . \tag{9}$$

We note that if $P(t)$ is taken independent of t in the interval $0 \leqq t \leqq \delta$, equation (9) is satisfied. This suggests that we seek a function of x and δ alone, which would satisfy (5). Moreover, such a function must also satisfy (6), since the first integral on the right-hand side of (6) involves $P(y)$ in the interval $(0, \delta)$.

Let us first compute $P(0)$. The first integral on the right side of (5) vanishes, and we have

$$P(0) = \int_{\delta}^{\infty} xP(y)\,dy . \tag{10}$$

But the form of $P(y)$ for $y > \delta$ is given by (8). Hence

$$P(0) = Ae^{-x\delta}. \tag{11}$$

Similarly, we compute $P(\delta)$ and find it to be also equal to $Ae^{-x\delta}$. This suggests that $P(t)$ might be Ae^{-xt} in the interval $0 \leqq t \leqq \delta$, and it remains to compute A. This quantity can be easily determined by the normalization of $P(t)$. We have

$$\begin{gathered} \int_0^{\infty} P(t)\,dt = \int_0^{\delta} Ae^{-xt}dt + \int_{\delta}^{\infty} Ae^{-xt}dt = 1; \\ \delta Ae^{-x\delta} + Ae^{-x\delta}/x = 1, \end{gathered} \tag{12}$$

whence

$$A = xe^{x\delta}(x\delta + 1)^{-1}. \tag{13}$$

Therefore, we guess the solution of the integral equation (5) and (6) to be

$$\begin{aligned} P(t) &= x(x\delta + 1)^{-1} \quad \text{for} \quad 0 \leqq t \leqq \delta; \\ P(t) &= x \operatorname{Exp}\{-x(t-\delta)\} \cdot (x\delta - 1)^{-1} \end{aligned} \tag{14}$$

for $t > \delta$.

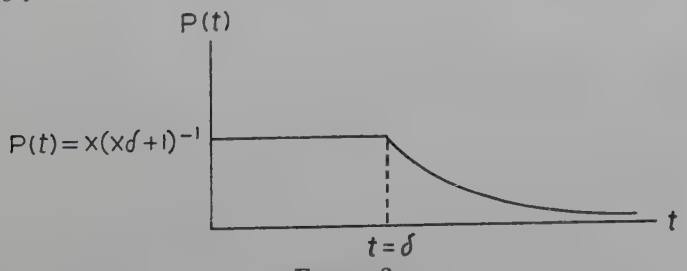

FIGURE 2

Substitution of (14) in (5) and (6) reduces the latter to identities. Hence, by the uniqueness of the solutions of (5) and (6), $P(t)$, as given by (14), is the function sought. The graph of $P(t)$ is shown in Figure 2.

We are now ready to compute the output intensity of an aggregate of neurons of the type N_3. If an arbitrary instant is chosen, the probability that N_2 has not fired for σ units prior to that instant is given by

$$Q(\sigma) = 1 - \int_0^\sigma P(t)\,dt. \tag{15}$$

There are two cases, $\sigma < \delta$ and $\delta < \sigma$. The first case seems biologically more plausible,† but for the sake of completeness, we shall treat both cases.

Case: $\sigma < \delta$.

A necessary condition for the firing of N_3 in an infinitesimal interval $(t, t + dt)$ is that it receives an impulse from N_1 and has not received one from N_2 for at least σ seconds prior to t. We must show that the condition is also sufficient. But the only thing that can prevent N_3 from responding to a stimulus received from N_1 which has not been "canceled" by a stimulus from N_2 is the possibility that the stimulus from N_1 has fallen within the refractory time of N_3. But if this happens, it means that N_3 has received two impulses from N_1 within less than δ seconds, because N_3 can be fired only by N_1. But this is impossible because of the refractory period of N_1. Therefore any stimulus coming from N_1 cannot fall within the refractory period of N_3, and the above-mentioned condition is sufficient for N_3 to fire. The independence of N_1 and N_2 now allows us to write down the probability that N_3 fires in any given infinitesimal interval dt, provided $\sigma < \delta$. This is

$$\begin{aligned} x_3 dt &= x(x\delta + 1)^{-1} Q(\sigma)\,dt = x(x\delta + 1)^{-1} \\ &\quad \cdot [1 - \sigma x(x\delta + 1)^{-1}]\,dt. \end{aligned} \tag{16}$$

The average frequency of firing of N_3 is obtained by dividing the right side of (16) by dt.

If the output is to have a maximum, then the equation

$$\frac{dx_3}{dx} = \frac{1}{(x\delta + 1)^2} - \frac{2\sigma x}{(x\delta + 1)^3} = 0 \tag{17}$$

†See, however, the remarks in this connection in IV (Rapoport, 1950d).

must have a solution for a positive value of x. Equation (17) indicates that the maximizing value of the input is given by

$$x^* = (2\sigma - \delta)^{-1}, \tag{18}$$

which is positive only if $\sigma > \delta/2$. There is thus a lower limit, namely, $\delta/2$, which σ must exceed in order for the aggregate of neurons of the type N_3 to exhibit a maximum in the output.

If we designate by x_1 the average frequency of N_1 (which by symmetry is also that of N_2), we can easily compute x_1, the value of x_1 which maximizes x_3. This turns out to be independent of δ and is given by

$$x_1^* = (2\sigma)^{-1} \quad \text{for} \quad \delta > \sigma > \delta/2. \tag{19}$$

The maximum value of x_3 is likewise independent of δ and is given by

$$x_3^* = (4\sigma)^{-1}. \tag{20}$$

Thus whenever the average frequency of the neurons N_3 is maximized, it is just one half the frequence of the neurons N_1 and N_2. We may also compute the limiting frequence of N_3 as x increases without bound. This does depend both upon δ and σ. Since $\operatorname{Lim}_{x\to\infty} x_1 = 1/\delta$, we have

$$\operatorname{Lim}_{x\to\infty} x_3 = 1/\delta - \sigma/\delta^2. \tag{21}$$

If $\sigma < \delta$, this is positive and approaches 0 as σ approaches δ (cf. IV).

Case: $\sigma > \delta$.

We now have

$$\begin{aligned}\int_0^\sigma P(t)\,dt &= \frac{\delta x}{x\delta + 1} + \int_\delta^\sigma \frac{x}{x\delta + 1} \operatorname{Exp}\Big\{-x(t-\delta)\Big\}\,dt \\ &= \frac{\delta x}{x\delta + 1} - \frac{\operatorname{Exp}\{x(\delta - \sigma)\} - 1}{x\delta + 1} \\ &= 1 - \frac{\operatorname{Exp}\{x(\delta - \sigma)\}}{x\delta + 1}.\end{aligned} \tag{22}$$

Then

$$Q(\sigma) = 1 - \int_0^\sigma P(t)\,dt = \operatorname{Exp}\left\{ x(\delta - \sigma) \right\} (x\delta + 1)^{-1} \tag{23}$$

and

$$x_3 = x \operatorname{Exp}\{x(\delta - \sigma)\}(x\delta + 1)^{-2}. \tag{24}$$

Since $\delta - \sigma < 0$, it is evident that $\lim_{x\to\infty} x_3 = 0$.

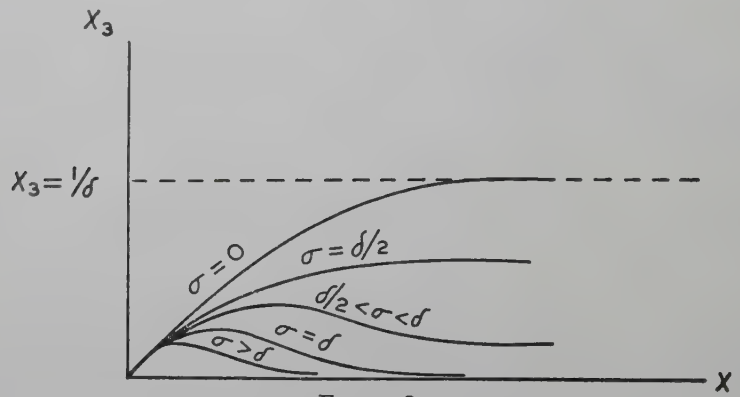

FIGURE 3

To obtain the maximizing input, we set

$$\frac{dx_3}{dx} = \frac{(x\delta+1)\,[\operatorname{Exp}\{x(\delta-\sigma)\}(1+x\delta-x\sigma)] - 2\delta x \operatorname{Exp}\{x(\delta-\sigma)\}}{(x\delta+1)^4} = 0 \tag{25}$$

and after simplifications obtain

$$x^2(\delta^2 - \delta\sigma) - x\sigma + 1 = 0, \tag{26}$$

which yields

$$x^* = \frac{\sigma \pm \sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2}}{2\delta(\delta - \sigma)}. \tag{27}$$

Discarding the negative root, we obtain for the maximizing input

$$x^* = \frac{\sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2} - \sigma}{2\delta(\sigma - \delta)}, \quad (\sigma > \delta). \tag{28}$$

Note that the limit of x^* as σ approaches δ is $1/\delta$, as should be the case.

The determination of x_1^* and x_3^* is straightforward. We have

$$x_1^* = (\sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2} - \sigma)\,(\sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2} + \sigma - 2\delta)^{-1} \quad (29)$$

$$x_3^* = \frac{2(\sigma - \delta)\,(\sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2} - \sigma)}{(\sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2} + \sigma - 2\delta)^2}\,\mathrm{Exp}\,\frac{\sigma - \sqrt{\sigma^2 + 4\delta\sigma - 4\delta^2}}{2\delta}. \quad (30)$$

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CONTRIBUTION TO THE PROBABILISTIC THEORY OF NEURAL NETS: IV. VARIOUS MODELS FOR INHIBITION†

ANATOL RAPOPORT
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

Input-output formulas are derived for a neuron upon which converge single axones of two other neurons, which are subjected to a Poisson shower, where a number of different assumptions are made concerning the mechanism of inhibition.

In one assumption so-called "bilateral pre-inhibition" is considered. That is to say, both neurons N_1 and N_2 may excite N_3, but if the stimulus of one of them follows within a certain interval σ of the other, the second stimulus is not effective. This model is essentially no different from that involving two excitatory neurons acting upon a neuron having a refractory period.

Another mechanism considered involves so-called "pre-and-post" inhibition, in which if two stimuli from N_1 and N_2 fall within σ, *both* are ineffective. This case being mathematically much more involved than the preceding, an approximation method is used for deriving the input-output formula.

It is assumed in the so-called two-factor theory (e.g., Rashevsky, 1948; Householder and Landahl, 1945) that neuroelements may be specifically characterized as either excitatory or inhibitory, that is to say, the action of some terminal bulbs enhances the probability of firing of the cell body they impinge upon, while the action of others, on the contrary, tends to counteract this. The discussions based on the two-factor theory have usually been so worded as to imply that the respective actions of excitatory and inhibitory bulbs are essentially different.

Actually it is not necessary to assume this. W. S. McCulloch has pointed out (in a lecture, May 25, 1950) that it is possible to account for a specific inhibitory effect of one *neuron* upon another without postulating any specific inhibitory action of its end bulbs.

Consider the net shown in Figure 1. The neuron N_4 fires if simultaneously excited by N_2 and N_3 (that is, the period of latent addition is zero). If N_5 does not fire, the firing of N_1 is sufficient to fire N_4 via N_2 and N_3. If, however, N_5 fires within δ units prior to N_1,

†Previous papers of this series are denoted by I, II, and III in this paper.

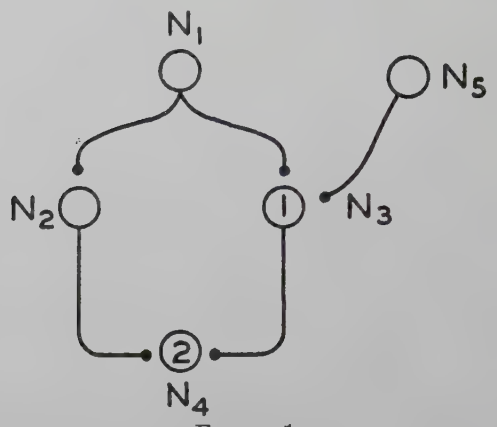

FIGURE 1

where δ is the refractory period of N_3, then N_3 will not be able to respond to the stimulation by N_1, since the stimulus from N_1 arrives at N_3 within its refractory period. But since N_4 has threshold 2, N_2 is not sufficient to fire it. Essentially, then, N_5 does produce an inhibitory effect upon N_4 with a period of latent inhibition equal to δ, the refractory period of N_3.

If the refractory period of N_4 does not exceed δ, it is seen from this model that it may be useful to consider cases in which the period of latent inhibition exceeds the refractory period (cf. III).

Note that although the McCulloch model obviates the necessity of postulating specific inhibitory action at the end bulbs, it is nevertheless *formally* equivalent to the two-factor theory models. The computation of the input-output curve of N_4 with respect to the frequency of Poisson-distributed stimuli impinging upon N_1 and N_5 would proceed exactly as in III where σ would now be the refractory period of N_3 and δ the refractory period of N_4.

As in the case discussed in III, it follows from the model represented in Figure 1 that

1) the action of neurons is specific (either inhibitory or excitatory, e.g., N_1 *always* tends to excite N_4, while N_5 *always* tends to cancel the action of N_1);

2) to effect inhibition, the inhibitory stimulus must arrive *before* the excitatory stimulus which it is to "cancel."

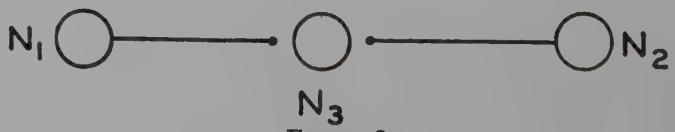

FIGURE 2

It is, however, possible to generalize the picture still further by dropping assumptions 1) and 2). In fact, let neurons N_1 and N_2 converge upon N_3 as in Figure 2.

The firing equation assumed in III was

$$N_3(t) \cdot\equiv\cdot N_1(t-1) \cdot [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1)}] \cdot [\tau_2 < \sigma \cdot\equiv\cdot \overline{N_2(t-\tau_2-1)}. \tag{1}$$

But we may assume instead of equation (1) any of the following equations

$$N_3(t) \cdot\equiv\cdot N_1(t-1)\ [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1}] \cdot [\tau_2 < \sigma \cdot\equiv\cdot \overline{N_2(t+\tau_2-1)}]\,, \tag{2}$$

$$N_3(t) \cdot\equiv\cdot \{N_1(t-1) \cdot [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1)}] \cdot [\tau_2 < \sigma \cdot\equiv\cdot \overline{N_2(t-\tau_2-1)}]\}\,,$$

$$\mathrm{v}\,\{N_2(t-1) \cdot [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1)}] \cdot [\tau_2 < \sigma \cdot\equiv\cdot \overline{N_1(t-\tau_2-1)}]\}\,, \tag{3}$$

and still others where the expressions $\overline{N_i(t-\tau_2-1)}$ are replaced by $\overline{N_i(t+\tau_2-1)}$ or by $\overline{N_i(t\pm\tau_2-1)}$; $(i=1,2)$. It is assumed throughout that $\sigma < 1$, i.e., does not exceed the synaptic delay.

Equation (3), for example, implies that either N_1 or N_2 may excite N_3, but that either N_1 or N_2 may cancel the stimulus of the other, provided its stimulus arrives within σ (period of latent inhibition) units before the stimulus of the other. Replacing $\overline{N_i(t-\tau_2-1)}$ by $\overline{N_i(t\pm\tau_2-1)}$ implies that inhibition can occur if the inhibitory stimulus arrives simply *within* σ units of the excitatory stimulus and may either precede or follow it. The term $\overline{N_i(t+\tau_2-1)}$ requires that the inhibitory stimulus *follow* the excitatory one (provided, of course, that it arrives within the synaptic delay, which is guaranteed by the condition, $\sigma < 1$).

Underlying each of the equations (1)—(3) and the alternatives suggested above is an interpretation of its possible "physical significance." Thus the model described by equation (2) may be the following: Firing takes place due to an accumulation of "excitatory substance." Inhibition consists in the destruction or neutralization of the excitatory substance but not by an accumulation of a specific "inhibitory substance." Thus, to be effective, the inhibitory stimulus must *follow* the excitatory one, since prior to the excitatory stimu-

lus there is no excitatory substance to neutralize.

Taking equation (3) with the terms $\overline{N_i(t - \tau_2 - 1)}$ replaced by $\overline{N_i(t \pm \tau_2 - 1)}$, we may interpret it as follows. Firing takes place as a result of an increased concentration of ions (accumulating during the synaptic delay) within the cell body near the point of excitation. If the bulbs of N_1 and N_2 are at such a distance from each other as to "compete" for the available ions, the (nearly) simultaneous activity of both neurons may preclude a sufficient concentration at either connection. Thus each neuron inhibits the other and the "inhibitory" stimulus may precede or follow the other.

Which of the equations (if any) is the most useful depends, of course, on the actual physical events associated with excitation and inhibition. In our formal treatment we will leave the question of physical significance open and will only work out the consequences of some of the assumptions. Equation (1) may be considered as representing "unilateral pre-inhibition," i.e., only N_2 inhibits and does so only if its stimulus arrives prior to that of N_1. Equation (3) represents "bilateral pre-inhibition" (both N_1 and N_2 may excite or inhibit, and the inhibitory stimulus must come before the excitatory). Entirely analogously we may represent unilateral and bilateral "post-inhibition" and unilateral and bilateral "pre-and-post-inhibition," which, to avoid a clumsy term, we shall designate as simply "inhibition."

Bilateral Pre-Inhibition or Simple Summation.

In the case of bilateral pre-inhibition N_3 can be fired by either N_1 or N_2 provided it had not fired for δ seconds previously and provided it had not been inhibited for σ seconds previously. The neuron N_3 remains unresponsive for δ units after firing. If $\delta > \sigma$, then, after the refractory period has passed, N_3 can be fired again by either N_1 or N_2. On the other hand if $\delta < \sigma$, then N_3 remains refractory for σ units after having been fired by N_1 or N_2. In either case, only one of the parameters (the greater of δ and σ) plays a part. The case of bilateral pre-inhibition thus reduces to one of simple summation of frequencies modified by a refractory period. That is to say, N_3 responds to a stimulus from either N_1 or N_2, provided the stimulus does not fall within the refractory period of N_3. For simplicity, the refractory periods of all three neurons will be taken equal to δ.

If N_3 were responding to two superimposed Poisson showers, each of frequency x, then the response of N_3 would simply be

$$f(2x) = \frac{2x}{2\delta x + 1} \tag{4}$$

in accordance with the considerations presented in I. However, the stimuli impinging upon N_3 from N_1 and N_2 are not Poisson-distributed, and we cannot simply take the sum of their frequencies for the input of N_3.

We shall first compute the function $p(\delta, t)$, the probability distribution of the time of the next firing of N_3, when the origin has been chosen at a moment δ units after a firing. From this the expected time of firing can be computed. Adding to this expected time, the refractory period δ, one obtains the expected time between two successive firings and hence the average frequency.

It is evident that $p(\delta, t)dt$ is the probability of receiving the *first* stimulus either from N_1 or from N_2 during the interval $(t, t+dt)$, it being known that a stimulus was received from one of the neurons at the instant $(-\delta)$.

Suppose for definiteness that the last stimulus was received from N_1. Then, since the refractory period of N_1 has passed, the probability distribution for the time of the next stimulus from N_1 will be given by xe^{-xt}, which is simply the probability distribution of the time of the next stimulus from the outside. Nothing, however, is known about the state of N_2 at our origin. Therefore, the distribution of the time of the next firing of N_2 must be given by equation (14) of III, i.e.,

$$\begin{aligned} &x(x\delta + 1)^{-1}, \quad \text{for} \quad 0 \leqq t \leqq \delta; \\ &x \operatorname{Exp}\{-x(t-\delta)\}\, x(x\delta + 1)^{-1}, \quad \text{for } t > \delta. \end{aligned} \tag{5}$$

Now the probability that the first stimulus after $t = 0$ received sitions may be considered mutually exclusive, since the probability that either this stimulus came from N_1, and no stimulus came from N_2 in the interval $(0, t + dt)$, or that it came from N_2 and no stimulus came from N_1 in the interval $(0, t + dt)$. The two propositions may be considered mutually exclusive, since the probability of simultaneous arrival of stimuli in the interval $(t, t + dt)$ is an infinitesimal of the second order. We thus obtain

$$p(\delta, t) = xe^{-xt}\left(1 - \int_0^t \frac{x\,dt}{x\delta + 1}\right) + \frac{x}{x\delta + 1}\left(1 - \int_0^t xe^{-xt}dt\right), \tag{6}$$

for $t \leqq \delta$;

$$p(\delta, t) = xe^{-xt}\left[1 - \int_0^{\delta} \frac{x\,dt}{x\delta + 1} - \int_{\delta}^{t} \frac{x \operatorname{Exp}\{-x(t-\delta)\}}{x\delta + 1}\,dt\right] + \frac{x \operatorname{Exp}\{-x(t-\delta)\}}{x\delta + 1}\left[1 - \int_0^{t} e^{-xt}\,dt\right] \tag{7}$$

for $t > \delta$.

After performing the indicated integrations, we get

$$p(\delta, t) = e^{-xt}\left[\frac{2x + x^2(\delta - t)}{x\delta + 1}\right], \quad \text{for} \quad t \leqq \delta; \tag{8}$$

$$p(\delta, t) = \frac{2x \operatorname{Exp}\{-x(2t-\delta)\}}{x\delta + 1}, \quad \text{for} \quad t > \delta. \tag{9}$$

For $t = \delta$, the two expressions (8) and (9) coincide.

To obtain the expected time of firing from our origin, we evaluate $\int_0^{\infty} tp(\delta, t)\,dt$. The integration is laborious but straightforward. In the interval $(0, \delta)$ we obtain

$$\int_0^{\delta} tp(\delta, t)\,dt = \frac{\delta(1 - e^{-x\delta})}{x\delta + 1}, \tag{10}$$

while in the interval (δ, ∞), we have

$$\int_{\delta}^{\infty} tp(\delta, t)\,dt = \frac{(2x\delta + 1)e^{-x\delta}}{2x(x\delta + 1)}. \tag{11}$$

Hence the expected time of the next firing from our origin, which, it will be recalled, was chosen δ units after firing, will be

$$E(\delta) = \frac{2x\delta + e^{-x\delta}}{2x(x\delta + 1)}. \tag{12}$$

Finally, the expected time of firing from a moment of firing is evidently

$$E(0) = E(\delta) + \delta = \frac{2x^2\delta^2 + 4x\delta + e^{-x\delta}}{2x(x\delta + 1)}. \tag{13}$$

The average frequency of x_3, being the inverse of the expected time between two successive firings, will then evidently be

$$x_3 = \frac{2x(x\delta + 1)}{2x^2\delta^2 + 4x\delta + e^{-x\delta}}. \tag{14}$$

It is seen that for very small values of x, x_3 behaves like $2x$, while for very large values, it approaches $1/\delta$ asymptotically, as should be the case.

An Approximation Method.

The problem of bilateral pre-inhibition, which, as we have seen, reduces to a simple summation of frequencies modified by the refractory period (assumed equal for all three neurons) can also be attacked by an approximation method. We shall use this method in the considerably more complicated case of bilateral inhibition (pre- and post-inhibition). Having solved the simpler pre-inhibition problem by both methods, we shall be able to estimate for that case the accuracy of the approximation method. As we shall see, the discrepancy does not exceed 8% in the simpler case. Hence, it is at least plausible that the method is useful in the closely related, but more involved, case of bilateral inhibition.

Consider all the stimuli received by N_3 from N_1 and N_2 (whether effective or not) distributed as points on the time axis. Because of the refractory period of N_3, only a fraction of these stimuli will be able to fire N_3. Let us now determine this fraction θ.

To do this, pick an arbitrary stimulus and ask what is the probability that it is "effective," that is, that it fires N_3. This is evidently the case if, and only if, an effective stimulus has not been received for δ units prior to it. To determine the probability that no effective stimulus has been received within a given interval of time, we must know the probability distribution of the time of its first occurrence from a given origin.

Essentially, the "exact" method above was based on the derivation of such distributions. In our approximation method, we suppose that the rectangular distribution which we have derived for the first occurrence of *any* stimulus within δ units of an arbitrarily chosen moment is simply modified by a constant factor to give the distribution of the first *effective* stimulus. This constant factor is evidently approximately θ.

We thus have the following equation for the probability that a given stimulus is effective.

$$\theta = 1 - \theta \int_0^{\delta} \frac{x\,dt}{x\delta + 1} = 1 - \frac{\theta \delta x}{x\delta + 1}, \tag{15}$$

which, solved for θ, gives

$$\theta = \frac{x\delta + 1}{2x\delta + 1}. \tag{16}$$

Multiplying this probability by the frequency of all stimuli received by N_3, that is, $2x(x\delta + 1)^{-1}$, we obtain for the approximate formula of the average frequency of N_3

$$\bar{x}_3 \sim \frac{2x}{2x\delta + 1} = \frac{2x(x\delta + 1)}{2x^2\delta^2 + 3x\delta + 1}. \tag{17}$$

The last expression of (17) has been put in this form to show its essential similarity to the "exact" formula (14). Note that (14) and (17) agree for both small and large values of x. Maximum discrepancy between them has been shown by S. Levinsohn (1951) not to exceed 8%.

Bilateral Pre-and-Post-Inhibition.

We shall now apply our approximation method to bilateral inhibition symbolized by the following equation:

$$\begin{aligned} N_3(t) \cdot\equiv\cdot \{ & N_1(t-1) \cdot [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1)}] \\ & \cdot [\tau_2 < \sigma \cdot\equiv\cdot \overline{N_2(t \pm \tau_2 - 1)}]\}, \\ \vee \{ & N_2(t-1) \cdot [\tau_1 < \delta \cdot\equiv\cdot \overline{N_3(t-\tau_1)}] \\ & \cdot [\tau_2 < \sigma \cdot\equiv\cdot N_1(t \pm \tau_2 - 1)]\}. \end{aligned} \tag{18}$$

That is to say, both N_1 and N_2 can excite N_3 and each can cancel the other's stimulus both before and after it is received. In other words, if stimuli from N_1 and N_2 arrive within the period of latent inhibition σ, *both* are canceled.

Again, as in the preceding example, consider all the stimuli received by N_3 from either source (whether effective or not) as a set of points distributed on the time axis. Clearly, the average density of these points will be $2x(x\delta + 1)^{-1}$. But only a fraction of the stimuli will be effective. We proceed to determine this fraction θ. We shall suppose that $\sigma < \delta/2$.

Note that an arbitrarily chosen stimulus received by N_3 is effective if, and only if, it is not preceded by an *effective* stimulus for δ units and is not preceded or followed by *any* stimulus for σ units. However, the two conditions are not independent, and the probability that both hold simultaneously cannot be obtained by multiplying the probabilities of each of the conditions together. Therefore, we introduce the probability $Q(\delta, 2\sigma)$ which is defined as the probability that no effective stimulus precedes a given moment, where *it is known* that no stimulus of any kind occurred for σ units before and after that moment. Now the probability that a given stimulus is effective can be obtained by taking the product of the following two probabilities: 1) the probability that no stimulus of any kind precedes or follows the stimulus in question within the interval σ; and, 2) the probability $Q(\delta, 2\sigma)$ defined above. As in the preceding case we again take a rectangular distribution for the first probability. Thus,

$$\theta = 1 - \frac{2x\sigma}{x\delta + 1} Q(\delta, 2\sigma). \tag{19}$$

It remains to compute $Q(\delta, 2\sigma)$. Essentially the computation involves the determination of $P(t)$ as in equations (5) and (6) of III, except that the origin is now not entirely arbitrary since it is known that no stimuli have been received for σ units before and after. The procedure will be equivalent to that followed in III if we choose our origin at $(-\sigma)$, measure time backwards, and suppose that no stimulus was received in the interval $(-2\sigma, 0)$.

Then $P(t)$, as given by the integral equations below, will represent the probability distribution of the time of the first impingement of an effective stimulus (actually the probability distribution of the time of the last firing)

$$P(t) = \int_{\delta - t}^{\delta} P(y)\, x \operatorname{Exp}\Big\{-x(t - \delta + y)\Big\} dy + \int_{\delta}^{\infty} P(y)\, x e^{-xt} dy; \tag{20}$$

for $t \leqq \delta - 2\sigma$,

$$P(t) = \int_{2\sigma}^{\delta} P(y)\, x \operatorname{Exp}\Big\{-x(t - \delta + y)\Big\} dy + \int_{\delta}^{\infty} P(y)\, e^{-xt} dy, \tag{21}$$

for $t > \delta - 2\sigma$.

Equations (20) and (21) yield solutions analogous to those obtained in III, namely,

$$P(t) = \frac{x}{x(\delta - 2\sigma) + 1}, \quad \text{for} \quad 0 \leqq t \leqq \delta - 2\sigma; \tag{22}$$

$$P(t) = \frac{x \operatorname{Exp}\{-x(t - \delta + 2\sigma)\}}{x(\delta - 2\sigma) + 1}, \text{ for } t > \delta - 2\sigma. \tag{23}$$

Since our origin was chosen at $(-\sigma)$ relative to the arbitrarily chosen stimulus, the probability that the first effective stimulus was received anywhere within δ units from $t = 0$ (it being known that none was received in the interval $(0, -\sigma)$), will be given by

$$\begin{aligned} \int_0^{\delta-\sigma} P(t) &= \frac{x(\delta - 2\sigma)}{x(\delta - 2\sigma) + 1} + \int_{\delta-2\sigma}^{\delta-\sigma} \frac{x \operatorname{Exp}\{-x(t - \delta + 2\sigma)\}}{x(\delta - 2\sigma) + 1} \\ &= \frac{x(\delta - 2\sigma)}{x(\delta - 2\sigma) + 1} + \frac{1 - e^{-x\sigma}}{x(\delta - 2\sigma) + 1}. \end{aligned} \tag{24}$$

From (24) we obtain $Q(\delta, 2\sigma)$, the probability that no effective stimulus was received in the interval $(-\delta, 0)$, it being known that no stimulus of any sort was received in the interval $(-\sigma, \sigma)$:

$$\begin{aligned} Q(\delta, 2\sigma) &= 1 - \theta \int_0^{\delta-\sigma} P(t)\,dt \\ &= 1 - \frac{\theta(x\delta - 2x\sigma + 1 - e^{-x\sigma})}{x(\delta - 2\sigma) + 1}. \end{aligned} \tag{25}$$

Substituting (25) into (19), we obtain the equation for θ, namely,

$$\theta = \left[1 - \frac{2x\sigma}{x\delta + 1}\right]\left[1 - \frac{\theta(x\delta - 2x\sigma + 1 - e^{-x\sigma})}{x(\delta - 2\sigma) + 1}\right], \tag{26}$$

which, solved for θ, gives

$$\theta = \frac{x\delta - 2x\sigma + 1}{2x\delta - 2x\sigma + 2 - e^{-x\sigma}}. \tag{27}$$

For $\sigma = 0$, the right side of (27) reduces to (16), which, of course, should be the case. Multiplying θ by the input intensity, $2x(x\delta + 1)^{-1}$, we finally obtain the approximate output intensity

$$\bar{x}_3 = \frac{2x(x\delta - 2x\sigma + 1)}{(x\delta + 1)(2x\delta - 2x\sigma + 2 - e^{-x\sigma})}. \tag{28}$$

If the accuracy of the approximation (28) is comparable with

that of (17), it can be used to derive the critical value of σ for which a maximum exists for x_3.

We note that for $\sigma = 0$, certainly no maximum exists, since there is no maximum for positive x of the expression (17). On the other hand if $\sigma = \delta/2$ (the maximum value for which our computations are valid), a maximum of (28) must exist, since in that case $\lim_{x\to\infty} \bar{x}_3 = 0$, while $d\bar{x}_3/dx > 0$ for $x = 0$.

To obtain σ^*, the critical value of σ, we should demand that $d\bar{x}_3/dx = 0$ have a positive real root. This involves the solution of a transcendental equation, which, however, can be easily obtained by graphical methods.

An immediate problem which suggests itself in connection with the various models proposed for inhibition is to compare the critical values of σ which insure the existence of a maximum in the input-output curve for each model, and the determination of the various limiting values of x_3 as x approaches infinity. The study of actual input-output curves should then indicate whether any of the proposed models can be taken as a hypothetical mechanism for excitation-inhibition phenomena.

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A REMARK ON THE DISTRIBUTION OF CONSERVATIVE QUANTITIES

N. RASHEVSKY
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

In line with a suggestion made in the recent book by the author, a distribution function is derived for the distribution in a social group of such conservative quantities for which every individual has a minimum requirement, this minimum requirement varying from individual to individual.

In chapter ix of our recent book (Rashevsky, 1950) it was pointed out that if a conservative quantity, that is, a quantity whose total amount remains constant, is distributed among the individuals of a population at random, the distribution curve is a simple exponential. The Number $N(x)\,dx$ of individuals who possess an amount of the quantity in question between x and $x + dx$ is of the form

$$N(x)\,dx = A\,e^{-ax}\,dx\,. \tag{1}$$

This is formally identical with the Boltzmann energy distribution. However, as remarked in *loc. cit.*, if x denotes incomes or wealth, then the expression (1) has meaning only for $x > \xi$, where ξ is the minimum amount of subsistence, since for $x < \xi$ an individual cannot exist. The distribution function of such quantities should therefore be represented by the curve shown in Figure 1 of chapter ix of *loc. cit.* That is, the curve is zero for $x < \xi$, and is given by $A \exp[-a(x - \xi)]$ for $x > \xi$. It thus exhibits a sharp angle at $x = \xi$.

Actual distribution curves of incomes, while roughly similar to that, do not exhibit the sharp angle. It was suggested in *loc. cit.* that this may be due to the circumstances that ξ varies from individual to individual, and that a smooth curve is obtained by a superposition of an infinite number of "angular" curves with different values of ξ.

In spite of the serious conceptual difficulties of this explanation, which were discussed in *loc. cit.*, we propose in this note to derive an expression for a smooth curve generated in the above-mentioned way.

Let $U(\xi)d\xi$ be the number of individuals whose ξ lies between ξ and $\xi + d\xi$. There is actually no human being for whom $\xi = 0$, so that the function $N(\xi)$ has a meaning only for $\xi > \Delta$, where Δ is the smallest possible value of ξ. It is also at least plausible that there is an upper limit Δ_1 of ξ, although the assumption of such an upper limit is not essential. The quantity x is assumed to be distributed according to $A \exp[-a(x - \xi)]$ for each group of individuals characterized by a given ξ, and in each group $x > \xi$. Hence the resulting distribution function $F(x)$ is given by

$$F(x) = A \int_{\Delta}^{x} U(\xi)\, e^{-a(x-\xi)}\, d\xi = A\, e^{-ax} \int_{\Delta}^{x} U(\xi)\, e^{a\xi}\, d\xi . \tag{2}$$

If an upper band Δ_1 for ξ exists, then for $x > \Delta_1$

$$F(x) = e^{-ax} \int_{\Delta}^{\Delta_1} U(\xi)\, e^{a\xi}\, d\xi . \tag{3}$$

Thus for $x > \Delta_1$ $F(x)$ in this case is a simple exponential of the same form as (1).

We may choose for $U(x)$ an inverted parabola

$$U(\xi) = B\left[(\xi - \Delta) - \frac{(\xi - \Delta)^2}{\Delta_1 - \Delta} \right]. \tag{4}$$

In this case $U(x)$ is zero for $\xi = \Delta$ and $\xi = \Delta_1$. If N_0 is the total population, then

$$\int_{\Delta}^{\Delta_1} U(\xi)\, d\xi = N_0 \tag{5}$$

and therefore

$$B = \frac{6N_0}{(\Delta_1 - \Delta)^2}. \tag{6}$$

This choice of $U(\xi)$ leads to rather cumbersome expressions. A simpler one is obtained by putting

$$U(\xi) = N_0\, b^2\, (\xi - \Delta)\, e^{-b(\xi-\Delta)}. \tag{7}$$

In this case there is no upper limit Δ_1.

Introducing (7) into (2) we find, putting $AN_0 = C$,

$$F(x) = Cb^2\left[\frac{1}{(b-a)^2}\, e^{-a(x-\Delta)} - \left(\frac{x - \Delta}{b - a} + \frac{1}{(b-a)^2} \right) e^{-b(x-\Delta)} \right]. \tag{8}$$

For $b >> a$ this reduces to

$$F(x) = C\{e^{-a(x-\Delta)} - [1 + b(x-\Delta)]\, e^{-b(x-\Delta)}\}. \qquad (9)$$

The function $F(x)$ is plotted for $a = 44 \times 10^{-5}$ dollar^{-1}, $b = 6 \times 10^{-3}$ dollar^{-1}, $C = 10^{7}$ individuals in Figure 1 below. For

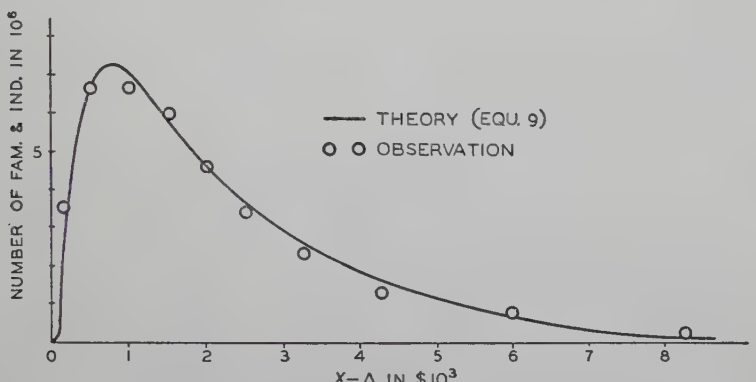

FIGURE 1

comparison, the circles show data from actual observations on the distribution of income, taken from page 253 of the *Statistical Abstracts for the United States,* 1946. In view of the objection raised in *loc. cit.* the agreement is not to be considered as too significant. It emphasizes, however, the need for a study suggested at the end of chapter ix of *loc. cit.*

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OUTLINE OF A MATHEMATICAL BIOLOGY OF LEADERSHIP

N. RASHEVSKY
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

Leadership, whether executive, political or any other type, is connected with the achievement of some goal by the social group through an appropriate organization of that group. From this point of view different leadership ranks in a group would be assigned to individuals according to their ability to organize the group for the purpose of reaching a specific goal. The situation is actually complicated by the circumstance that an individual may have the necessary ability but may not like the responsibility connected with the leadership, or vice versa. Also, he may not be interested in the goal. The suggested mathematical approach is to consider that the satisfaction of an individual is a function of his leadership rank, of the goal, and of several other parameters. If each individual tends to adjust his position in society so as to maximize his own satisfaction, this condition gives us the equations which determine the leadership rank of each individual. It is found that, in general, the rank of an individual depends not only on his ability, but on the abilities of all other individuals. The method enables us to calculate the distribution function of abilities among individuals of a given rank, and leads to results which allow, in principle, experimental verification.

In our previous studies of mathematical biology of social behavior (Rashevsky, 1948; 1950) we discussed possible mechanisms of change of behavior of a social group as a whole. For the particular case of only two possible mutually exclusive behaviors, A and B, we considered that the tendency, ϕ, toward one or another of those behaviors was distributed continuously within the population. Individuals with $\phi = 0$ have no preference toward either A or B. Individuals with $\phi > 0$ prefer A, those with $\phi < 0$ prefer B. Individuals with high absolute values of ϕ act for the most part according to their innate tendency ϕ, and their behavior is very little affected by imitation. Individuals with small absolute values of ϕ are, on the contrary, acting primarily by imitation. It was shown (Rashevsky, 1950; Landau, 1950; Landahl, 1950) that owing to imitation the majority of a social group will, in general, choose either behavior A or behavior B, even though the distribution of ϕ is symmetric with respect to $\phi = 0$. Which of the two behaviors is adopted is a matter of chance.

Changes in the behavior of a social group occur when the dis-

tribution function of ϕ changes so that the average value $\bar{\phi}$ of ϕ shifts sufficiently in one direction or another. In this picture there is no manner in which the single individual can influence the behavior of the group. This point of view leads to historical determinism which is explicit in our previous studies. Yet, within limits, individuals do affect the course of some events and do control the behavior of more or less large social groups. How do these cases fit into the above-mentioned theory of social behavior?

If a social group adopts a particular behavior; for example, A, we may perhaps consider that the few individuals with the highest positive value of ϕ will control the situation and will be the leaders. Such a picture, however, is too oversimplified, and does not take into account the following situation: There is a difference between the adoption of a behavior pattern and the ability to have this pattern properly organized. For instance, society may adopt a behavior pattern consisting of belligerent activities. It will extol militarism and begin aggressive wars. But it may not possess any military leaders of sufficient talent to insure victory in such wars. Or the accepted behavior pattern may be that of peaceful production of some goods. But in the absence of individuals who can properly organize such a production, no beneficial results are obtained.

Individual leadership pertains not to the general behavior pattern, but to its internal organization with a definite purpose in mind. In a sense, any theory of individual leadership should be developed on the basis of a general theory of biological and social organization (Rashevsky, 1946). Since at least some of the activities of mankind are purposeful, therefore mankind has organized certain hierarchies of leadership to expedite those purposes. These hierarchies are different in nature from the more general hierarchies studied previously (Rashevsky, 1950) which are established due to biological mechanisms in which purpose does not need to play any part.

Viewed from this angle, it would seem at first that the leadership of an individual will be determined by his ability to organize and direct certain tasks, and that in this hierarchy of leadership the top leader will be the one with greatest abilities, the next one with next best abilities, and so forth. That such is not actually the case is shown by everyday life, especially in the field of political leadership. Mediocrities occur also in leading positions in industries and other private organizations.

If we look for another plausible factor which may determine the leadership rank of an individual, we find that such a factor may be

the desire of the individual for leadership. A person may have all the abilities necessary to organize a certain group task. But the responsibilities and troubles connected with such organizational work may be very unpleasant to him, and he will not want a position of leadership. On the other hand, an individual of mediocre abilities may crave a leading position to such an extent that he may obtain it by sheer vociferation. This sounds ludicrous, but it actually occurs.

Another factor which must be considered is the desire of individuals for a particular goal which can be achieved by organization. An individual with high organizational abilities may be quite indifferent toward that goal. Another individual with lesser abilities may have a very strong desire for that goal. If most of the group desires that goal too, the less capable enthusiastic individual may become a leader, rather than the more capable indifferent one.

In dealing with problems of motivation, is it useful to introduce the concept of satisfaction function (Rashevsky, 1948; Rapoport, 1947a,b). Therefore we shall introduce it here.

The satisfaction function S_i of the ith individual in a group is, in the present case, a function of his leadership rank r_i and of the measure x of the achievement of the desired goal. The rank r_i may be an integer, assigned to each individual, thus: first in rank, second in rank, etc. Or it may be a continuous quantity, measured on an appropriate psychophysical scale (Rashevsky, 1950). The quantity x may represent anything, for example, the total industrial production by the group, the number of schools, or the quality of schools measured on an appropriate scale. The quantity x is the same for all individuals of the group, but the desire for it varies from person to person.

The satisfaction S_i may, in general, contain a number of parameters $\lambda_k^{(p)}$, where the subscript refers to the individual, the superscript to the nature of the parameter. Thus $\lambda_1^{(1)}$, $\lambda_2^{(1)}$, $\lambda_3^{(1)}$ $\cdots$ are values of the same parameter for different individuals, while $\lambda_1^{(1)}$, $\lambda_1^{(2)}$ $\cdots$ are values of different parameters for the same individual. The satisfaction S_i of the ith individual may, in general, depend not only on the $\lambda_i^{(p)}$'s, but also on the values $\lambda_k^{(p)}$ of the parameters for other individuals. Denoting by N_0 the total number of individuals, and by m the number of different parameters λ, we thus have

$$S_i = S_i(r_i, x; \lambda_k^{(p)}); k = 1, 2 \cdots N_0; p = 1, 2 \cdots m. \qquad (1)$$

Each individual may act either egoistically or altruistically, as well as intermediately (Rashevsky, 1950). In the first case each in-

dividual tries to maximize his own satisfaction by trying to choose the appropriate r_i. We thus have

$$\frac{\partial S_i}{\partial r_i} = 0, \tag{2}$$

together with the other standard conditions for a maximum.

Different individuals have different ranks r_i of responsibility and importance in an organization. The quantity x depends on the abilities of all the individuals concerned, as well as on the corresponding ranks r_i. If two individuals of different abilities interchange their leadership ranks, x will change. The quantity x may, in general, also be a function of the $\lambda_k^{(p)}$'s. Thus we have, with $k = 1, 2 \cdots N_0$; $p = 1, 2 \cdots m$:

$$x = f(a_1, a_2, \cdots, a_{N_0}; r_1, r_2, \cdots, r_{N_0}; \lambda_k^{(p)}). \tag{3}$$

Introducing (3) into (1), and then the latter into (2) we find N_0 equations for the determination of the N_0 values r_i. From them we find:

$$r_i = U_i(a_1, \cdots, A_{N_0}; \lambda_k^{(p)}); \quad k = 1, 2 \cdots N_0; \quad p = 1, 2 \cdots m. \tag{4}$$

If r_i is an integer by definition, then the integers nearest to the values given by equation (4) are to be taken.

Thus we see that the leadership rank of an individual depends not only on his own abilities, but on the abilities of all other individuals.

If each individual acts altruistically (Rashevsky, 1950), he will try to maximize $S = \sum_i S_i$. We then have instead of equations (2), the following one:

$$\frac{\partial S}{\partial r_i} = 0. \tag{5}$$

Introducing (3) into (5), we again find N equations for the determination of the N ranks r_i. Similarly, the most general case (Rashevsky, 1950) is treated in which the ith individual maximizes the linear form

$$k_{i1} S_1 + k_{i2} S_2 + \cdots + k_{iN_0} S_{N_0}. \tag{6}$$

If S_i depends on r_i only, then $\partial S/\partial r_i = \partial S_i/\partial r_i$ and egoistic and altruistic behaviors lead to the same result. It is, however, quite natural to assume that S_i depends not only on r_i but also on the

other r_k's. A person may be pleased or displeased to see a particular person occupy a definite rank. In such cases egoistic and altruistic behaviors lead, in general, to different results. The comparative study of the different behaviors offers an interesting problem.

We shall illustrate the above general method on two special cases. First let us put

$$S_i = \alpha_i \log (r_i + x) - \beta_i r_i \ , \tag{7}$$

where α_i and β_i are parameters.

Without loss of mathematical generality we may put $\beta_i = 1$, though psychologically it makes a difference whether an individual likes the quantity r more, or dislikes the effort to get that quantity r less.

In choosing the function f we must consider two possibilities. The rank may be measured in relative units. The individual who has the highest rank is relatively the most important and responsible one. In this case, f must be a homogeneous function of zeroth degree in the r_k's, because increasing all the r_k's n times does not change anything, and leaves x, and hence f, unchanged. On the other hand, we may consider absolute rank. This may, if convenient, be normalized so as to vary within the range 0, 1. An individual with rank 0.9 is not only relatively more responsible than an individual with rank 0.7, but actually has greater power. In a group in which the highest possible rank is 0.7, the highest ranking individual has less executive powers than an individual with rank 0.8 in another group, though in that other group the highest rank may be even higher, say, 0.95.

We shall consider here only the case of absolute rank. In that case we may choose the function f in equation (3) so that

$$x = \sum_i a_i r_i . \tag{8}$$

This is a plausible enough assumption. It means that the higher the ability of an individual with a given rank, the better the result. More generally we may put:

$$x = \sum_i a_i \alpha_i r_i \tag{9}$$

or

$$x = \sum_i (a_i + \alpha_i) r_i . \tag{10}$$

Let us choose expression (9) as an example. Introducing it into expression (7), and then the latter into (2) we find

$$\frac{\alpha_i(1+a_i\,\alpha_i)}{r_i+\sum_k a_k\,\alpha_k\,r_k}-1=0. \tag{11}$$

This gives

$$r_i=\alpha_i(1+a_i\,\alpha_i)-\sum_k a_k\,\alpha_k\,r_k. \tag{12}$$

The expression $\sum_k a_k\,\alpha_k\,r_k$ on the right side which is nothing other than x, does not depend on i. It is the same for all individuals, and may be treated as a constant, independent of i. Hence equation (12) may be written

$$r_i=\alpha_i(1+a_i\,\alpha_i)-x\,. \tag{13}$$

Introducing (13) into (9) we find:

$$x=\sum_k a_k\,\alpha_k[\alpha_k(1+a_k\,\alpha_k)-x]\,, \tag{14}$$

or

$$x=\frac{\sum_k a_k\,\alpha_k{}^2(1+a_k\,\alpha_k)}{1+\sum_k a_k\,\alpha_k}. \tag{15}$$

Hence, introducing (15) into (13), we find:

$$r_i=\alpha_i(1+a_i\,\alpha_i)-\frac{\sum_k a_k\,\alpha_k{}^2(1+a_k\,\alpha_k)}{1+\sum_k a_k\,\alpha_k}. \tag{16}$$

For very large values of N_0 we may substitute integrals for the sums. The rank r then becomes a function of the continuously distributed a and α, and is itself a continuous quantity. Let $N_1(a)$ and $N_2(\alpha)$ be the respective distribution functions of a and α in the social group, and let a also be normalized so as to vary from 0 to 1. Then:

$$r(a,\alpha)=\alpha(1+a\alpha)-\frac{N_0\int_0^1 a\alpha^2(1+a\alpha)\,N_1(a)\,N_2(\alpha)\,da\,d\alpha}{1+N_0\int_0^1 a\alpha\,N_1(a)\,N_2(\alpha)\,da\,d\alpha}. \tag{17}$$

If both $N_1(a)$ and $N_2(\alpha)$ are normal distributions with sufficiently small standard durations σ_1 and σ_2, and with medians α^* and α^*

near $\alpha = \frac{1}{2}$ and $\alpha = \frac{1}{2}$, then the integrals in (17) may be evaluated with good approximation by extending the range of integration from $-\infty$ to $+\infty$. Then r becomes a function of a, α, of the standard deviations σ_1 and σ_2, and of the medians a^* and α^*.

As another example we choose:

$$S_i = \alpha_i \log r_1 - r_1 + \beta \log x , \tag{18}$$

with either

$$x = \sum_k a_k \alpha_k \beta_k r_k, \tag{19}$$

or

$$x = \sum_k (n a_k + \xi \beta_k) r_k. \tag{20}$$

Using expression (19) we find now from equations (2):

$$\frac{a_i}{r_i} - 1 + \frac{a_i \alpha_i \beta_i^2}{\sum_k a_k \alpha_k \beta_k r_k} = 0, \tag{21}$$

or, remembering (19),

$$r_i = \frac{x a_i}{x - a_i \alpha_i \beta_i^2}. \tag{22}$$

Introducing (22) into (19) we find the following equation for the determination of x:

$$\sum_k \frac{a_k \alpha_k^2 \beta_k}{x - a_k \alpha_k \beta_k^2} = 1. \tag{23}$$

If N_0 is very large, and if $N_3(\beta)$ is the distribution function of β, while $N_1(a)$ and $N_2(\alpha)$ have the same meaning as before, then we may write instead of (23):

$$N_0 \int_0^1 \frac{a\alpha^2 \beta N_1(a) N_2(\alpha) N_3(\beta) da d\alpha d\beta}{x - a \alpha \beta^2} = 1. \tag{24}$$

In general this is a transcendental equation in x, and cannot be solved in closed form.

Consider now expression (17). It is, as we have seen, of the form:

$$r(a,\alpha) = F(a, \alpha; \sigma_1, \sigma_2, a^*, \alpha^*), \tag{25}$$

where the parameters σ_1, σ_2, a^* and α^* are characteristic constants for a given social group. Consider a specified constant rank r. The equation

$$r = F(a, \alpha; \sigma_1, \sigma_2, a^*, \alpha^*) \tag{26}$$

determines a curve in the a, α plane.

Omitting, for brevity, the constant parameters, and retaining only r, we may write the equation of the curve as follows:

$$\alpha = V(a, r). \tag{27}$$

Put

$$\frac{\partial V(a,r)}{\partial a} = V_a(a,r); \quad \frac{\partial V(a,r)}{\partial r} = V_r(a,r). \tag{28}$$

If instead of r we consider a value $r + dr$, we find instead of the curve (27), an infinitesimally near curve, whose distance from the curve (27) in the direction of the normal is

$$d\nu = \frac{V_r(a,r)}{\sqrt{1 + [V_a(a,r)]^2}}\, dr. \tag{29}$$

The length of the element ds of the curve is given by

$$ds = \sqrt{1 + [V_a(a,r)]^2}\, da, \tag{30}$$

so that an element $ds\, d\nu$ of area between the two infinitesimally near curves is equal to:

$$ds\, d\nu = V_r(a,r)\, da\, dr. \tag{31}$$

We may now ask for the relative probability of a given value of a in the strip between the two curves. This will give us the distribution of a for individuals whose rank is between r and $r + dr$. This is, in general, different from the distribution of a in the whole population.

The relative number of individuals with a and α lying within the area $da\, d\alpha$ around the point a,α is

$$N_1(a)\, N_2(\alpha)\, da\, d\alpha. \tag{32}$$

If we confine the area $ds\, d\nu$ to the infinitesimal strip discussed above, α is fixed by (27) for a given a, and we must substitute $V(a, r)$ for α, while for $da\, dx$ we substitute $ds\, d\nu$ as given by (31). We thus

obtain for the distribution function of a for a given interval $(r, r + dr)$

$$N_r(a) = N_1(a)\, N_2[V(a, r)]\, V_r(a, r)\, da\, dr. \qquad (33)$$

This is the relative frequency of individuals with an a between a and $a + da$, if their rank is between r and $r + dr$.

If instead of expression (17) we have the expression (22) in which there are two parameters, α and β, we can use the same procedure, referring now not to a curve, but to a surface.

If we assume a given expression for S, say, either (7) or (18), or else some still different one, we can in principle determine the distribution function $N_1(a)$, $N_2(a)$, etc. for the whole population. Then for every assumed form of S we obtain a definite expression for $N_r(a)$, which again in principle can be checked. Thus the assumption of a particular form of S may be verified.

If the rank r is very high, then there may be only one, or very few, persons in that rank. Expression (33) then gives us the relative incidence in time of a given value of ability a in leaders of such a high rank.

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AN APPLICATION OF THE THEORY OF NEURAL NETS TO THE STUDY OF MOTION SICKNESS

H. D. LANDAHL
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

The results from a study by M. F. Morales on the labyrinthine response to motion are used, together with results of neural net theory, to enable one to calculate, in terms of a small number of parameters, the percentage of individuals who have motion sickness when exposed to rather general patterns of motion which may vary with time.

In a paper by M. F. Morales (1946), a theory has been discussed which makes it possible to estimate with some success the effectiveness of the spatial and temporal pattern of a given periodic motion in producing motion sickness. The present note is an analytic treatment of the dose-response problem for this phenomenon.

Let the average frequency of the action spikes produced by the labyrinthine receptors during the cycle of the motion be denoted by $\nu - \nu_0$, where ν_0 is the value in the absence of motion. The value of ν (σ of Morales, 1949) shall be considered to be given in some arbitrary units by means of a mechanism such as that considered by M. F. Morales (1946) from a knowledge of the form of one complete cycle of the imposed wave. For any given pattern, ν would be proportional to the greatest value of the acceleration in any cycle. If ε and j are measures of excitatory and inhibitory factors at some center which produces a particular response under observation, then we may expect the following relations to hold, if the simplest possible connections from the labyrinthine receptors are postulated (Rashevsky, 1948):

$$\frac{d\varepsilon}{dt} = A\nu^n - a\varepsilon, \tag{1}$$

$$\frac{dj}{dt} = B\nu^n - bj, \tag{2}$$

where A, a, B, b, and n are constants, n being close to one. The

term v^n is introduced to allow for a possible non-linearity between the quantity E (Rashevsky, 1948, p. 357) and v.

We next introduce the rather reasonable postulate that, for the effect to be produced, $\varepsilon - j$ must exceed the threshold value h which is distributed over the population according to the distribution function $p(\sigma^{-1} \log h/\bar{h})$ where $\bar{h}$ is the median value of h, and where

$$p(\xi) = \frac{1}{\sqrt{2\pi}} e^{-\xi^2/2}. \tag{3}$$

We define

$$P(\alpha) = \int_{-\infty}^{\alpha} p(\xi)\, d\xi . \tag{4}$$

Thus for any value α, $P(\alpha)$ may be found from a table of the area under the normal error curve, e.g., if the values in the table range from zero to $\frac{1}{2}$ as the deviate moves from zero to infinity, $P(\alpha)$ is $\frac{1}{2}$ plus or minus the tabulated value, depending upon whether or not the value of α is positive or negative.

We shall now consider a few special cases.

1) Let the accommodative effect be negligible, i.e., $B << A$. Then we have the following result for the case of a constantly maintained motion producing a constant average v. The percentage in the population showing the effect under observation, e.g., nausea, will be given by $P_1(v, t)$:

$$P_1(v, t) = P\left[\frac{1}{\sigma} \log v^n (1 - e^{-at}) / \bar{v}^n \right] \tag{5}$$

$(v = \text{constant}, A >> B)$,

where $\bar{v}^n = a\bar{h}/A$, and $\bar{v}$ is thus the value of v which produces the effect in one-half of the population when the motion continues for a long time.

2) For the same conditions imposed upon the parameters as in the preceding case, consider a constant motion maintained for a time t_1. We then ask: How long will the effect last after t_1? After a time t_1 we have $\varepsilon - j$ given by

$$\varepsilon(t_1) - j(t_1) = \frac{A}{a} v^n (1 - e^{-at_1}), \tag{6}$$

so that after t_1 we have, if t is measured from t_1,

$$\varepsilon - j = \frac{A}{a} v^n \, (1 - e^{-at_1}) \, e^{-at}. \tag{7}$$

Now if $\varepsilon - j$ in equation (7) is set equal to $\bar{h}$, then t will be equal to some value $\bar{\tau}$, the median duration of the effect. Solving for $\bar{\tau}$ we obtain

$$\bar{\tau} = \frac{1}{a} \log \left[A v^n (1 - e^{-at_1}) / a \, \bar{h} \right]. \tag{8}$$

For sufficiently short exposures the median duration of the response is given by

$$\bar{\tau} = \frac{1}{a} \log A v^n \, t_1 / \bar{h}. \tag{9}$$

It may be that there is a minimum time τ_0 in which the effect can occur. Such a quantity may have to be introduced, especially when $\bar{\tau}$ is small.

3) If the accommodation cannot be neglected, then we obtain, for the case of a constantly maintained motion,

$$P_2(v, t) =$$

$$P \left[\frac{1}{\sigma} \log \frac{A v^n (1 - e^{-at}) / a - B v^n (1 - e^{-bt}) / b}{A \bar{v}^n \{1 - (B/A)^{a/(a-b)}\} / a - B \bar{v}^n \{1 - (B/A)^{b/(a-b)}\} / b} \right]. \tag{10}$$

Equation (5) or equation (10) then determines the percentage response as a function of the intensity and duration of the exposure

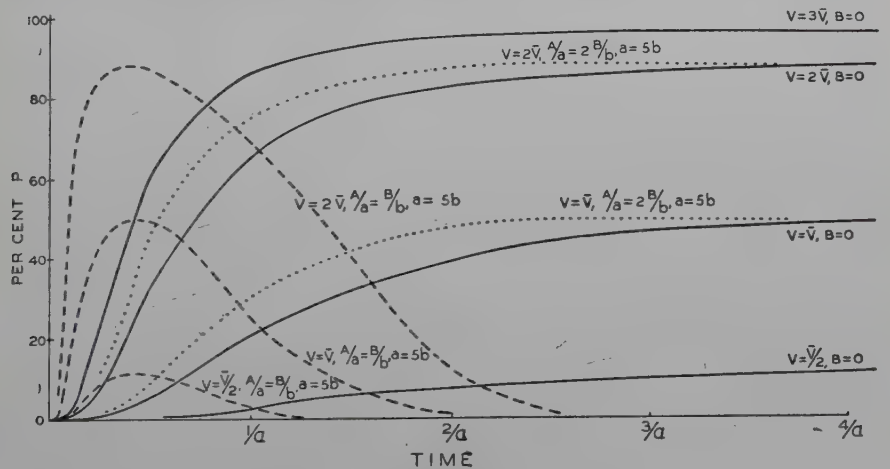

FIGURE 1

to the motion. The type of relation expected is illustrated in Figure 1 for several values of the parameters.

Evaluation of Parameters. First let the exposure times be sufficiently short so that $bt << 1$. Then for any time of exposure, the per cent of the population showing the effect should be approximately represented by expression (5), and thus a plot of the per cent against intensity of the motion (ν) on log-probability paper should result in roughly straight lines of slope $s = \sigma/n$, e.g.,

$$\sigma/n = \tfrac{1}{2} \log [\nu(84\%)/\nu(16\%)] .$$

For very short times of exposure and large intensities, one should find $\nu^n t =$ constant. Thus if P is plotted against t under these restrictions, approximately straight lines should be obtained, each with slope σ, e.g., $\sigma = \frac{1}{2} \log [t(84\%)/t(16\%)]$. Thus the ratio of σ to s is n. Now $\bar{\nu}$ is obtained, by interpolation if necessary, as that value of ν giving 50% response at the time when the response is maximum. If the accommodation ratio $(B/b)/(A/a)$ is a small fraction, or if $b << a$, $P_1(\bar{\nu}) = \frac{1}{2}$ for $t >> 1/a$, but $t < 1/b$.

If we set $P_1 = \frac{1}{2}$ then

$$\nu = \bar{\nu}/(1 - e^{-at})^{1/n} \quad (P = \tfrac{1}{2}). \tag{11}$$

Since $\bar{\nu}$ and n are estimated, this plot determines a approximately [cf. also equation (9)]. Estimation of $(B/b)/(A/a)$ and b is more difficult. Some idea of the value of these parameters might be obtained from recovery data. A more direct method would be the application of a test increase in intensity after various durations of known constant intensities (Landahl and Kahn, 1948). Another possible method would be that of application of periodic intensities, e.g., equal periods of motion and rest.

We will briefly mention some possible complications. It may be that a single time constant in the production of the observed effect is inadequate. This would mean that an ε_1 and ε_2 with corresponding A_1, a_1, A_2, a_2 must be introduced. This only introduces an additive term in the numerator and denominator of the argument of the function P. For example, if the accommodative effect were negligible, the expression for the percentage would be obtained by replacing B/b by $-A'/a'$ and b by a' in the argument of the function P. A more difficult problem arises, however, if we attempt to consider what occurs if parameters other than n are variable over the population. For example, some individuals might show considerable accommodation

while others show none. In this case we could perhaps neglect the variability of h and consider the relative accommodation to be distributed approximately normally over the population.

If the parameters have been estimated and the various predictions are approximately fulfilled, it would be possible to estimate the percentage of a population showing an effect, e.g., nausea, after the individuals' exposure to a known variable intensity $\nu(t)$. This is given by the expression

$$P_3\{\nu(t)\} =$$

$$P\left[\frac{1}{\sigma}\log\frac{\left\{Ae^{-at}\int_0^t \nu^n(t)\,e^{at}\,dt - Be^{-bt}\int_0^t \nu^n(t)\,e^{bt}\,dt\right\}}{A\bar{\nu}^n\{1-(B/A)^{a/(a-b)}\}/a - B\bar{\nu}^n\{1-(B/A)^{b/(a-b)}\}/b}\right].$$

If the expression is to give results after a possible decrease in P, i.e., the duration of the effect, then one may have to allow for the time τ_0 mentioned above, if it is appreciable. In principle, then, for any form of motion which may vary in time, the percentage at any given time showing the effect under observation can be calculated. To what extent these results can be made to agree with observation remains to be determined.

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SOME BIO-SOCIOLOGICAL ASPECTS OF THE MATHEMATICAL THEORY OF COMMUNICATION

N. RASHEVSKY
COMMITTEE ON MATHEMATICAL BIOLOGY
THE UNIVERSITY OF CHICAGO

In the first part of the paper a general discussion of the transmission of information through neural chains is given in terms of the Shannon-Weaver theory. It is pointed out that with the all-or-none law a single chain of neurons connected in series transmits one bit of information per signal. A set of N *independent* parallel chains transmits N bits per signal. If, however, the chains are interconnected, the amount of information is reduced. At the same time, however, the degree of coordination of the final neuromuscular reaction is increased. A relation between the maximum possible speed of a reaction and its degree of coordination is derived, and possible applications to spoken language are suggested. A general quantitative discussion of the relation between amount of information and amount of knowledge which an individual may obtain when confronted with the external world is made and a possible connection with new trends in logical thinking is pointed out.

In the second part transmission of information through "social chains" is discussed under certain special assumptions. An expression for the "social channel noise" in terms of the length of the channel is derived. Finally an expression is given for the amount of information transmitted from one individual to another in a social group of uniform density as a function of the physical distance between the two individuals.

The possible importance of the mathematical theory of communication for biology and sociology has been discussed in recent publications (Wiener, 1948; Shannon and Weaver, 1949). The purpose of this paper is to discuss some possible applications more specifically than has been done hitherto. In our discussion we shall follow the presentation and notations of C. Shannon and W. Weaver (1949). We shall first mention in a most general way some applications to the biology of the individual, then we shall study more specifically a problem of communication in social groups.

I. *The Individual.*

The overt, observable behavior of an individual is manifested in the vast majority of cases by a set of more or less complex motor reactions, which may be, and are frequently, used as channels of

communication. Perhaps the most important of those motor reactions is speech. The relatively more rare non-motor reactions are glandular secretions, such as sweating, salivating, and weeping. Of those only the latter is used as a channel of communication, and a rather important one at that. Confining ourselves to motor reactions does not entail any loss of generality.

Individual elementary motor reactions may be considered as signals or symbols, in the same sense as used by C. Shannon. They are caused either by external stimuli, or by internal stimuli or drives, which originate endogenically in the brain. In either case there is a transmission from the site of the stimulus to the motor ending. Different regions of the brain thus act as communication channels.

Assuming the general validity of the all-or-none law, we see that a neuron can either only fire or not fire. Thus it is capable of transmitting exactly one *bit* of information per signal (Shannon and Weaver, 1949). A simple chain of neurons in which all thresholds are sufficiently low, so that the firing of a preceding neuron always results in a firing of the subsequent one, also transmits exactly one bit per signal. If ν is the frequency of firing of each neuron in the chain, the rate of transmission C of information by such a simple chain is ν bits per second. The frequency ν is limited by the duration of the refractory phase of the neuron, and its highest possible value is of the order of magnitude of 10^2 sec^{-1}. Hence the maximum channel capacity C of a simple chain of neurons is about 10^2 bits sec^{-1}. If we have N independent parallel chains, then the channel capacity of such a system is of the order 10^2N bits sec^{-1}.

If the individual parallel chains are, however, interconnected by either excitatory or inhibitory links, the capacity of the channel is decreased. We shall illustrate this on the following examples.

Consider first two parallel chains. The amount of information transmitted *per signal* is, as we have seen, exactly two bits. Let the two chains, however, crossinhibit each other, so as to form the well-known circuit, studied by H. D. Landahl (1937) (cf. also Rashevsky, 1948). Let us first consider the case in which the random fluctuations of excitation and/or thresholds are absent. Then, with a proper choice of the parameters involved, the circuit can produce either one of the two reactions R_1 or R_2, or none, depending on the intensities S_1 and S_2 of the stimuli. We shall denote the latter case symbolically by $\bar{R}_1 \cdot \bar{R}_2$. This circuit cannot produce both reactions. Now we have three possible signals: R_1, when $S_1 - S_2 > h$; R_2, when $S_2 - S_1 > h$; and $\bar{R}_1 \cdot \bar{R}_2$, when $|S_1 - S_2| \leqslant h$. Hence the in-

formation per signal is now $\log_2 3 = 1.58$ bits. Now consider the general case in which fluctuations are present. We still have the same 3 possible signals, but their choices are not quite free. Even if we make $S_1 - S_2 > h$, there will be a probability P_w of reaction R_2 and a probability P_e of no reaction at all. Thus even if the choice between $S_1 - S_2 > h$, $S_2 - S_1 > h$, and $|S_1 - S_2| \leqslant h$ is equally probable, and equal to 1/3 for each choice, the signals R_1, R_2, and $\overline{R}_1 \cdot \overline{R}_2$ will not be equally probable. Hence the information per signal will be less than $\log_2 3$ (Shannon and Weaver, 1949). The actual amount of that information may be computed in the following way:

We shall first restrict further the assumption that the choice of either of the three inequalities

$$S_1 - S_2 > h; \quad S_2 - S_1 > h; \quad |S_1 - S_2| \leqslant h \tag{1}$$

is equally probable, by considering the case in which there is an equiprobable choice between 3 pairs S_1, S_2 of *fixed* values of S_1 and S_2, the three pairs satisfying respectively the three inequalities (1). Denote by P_1, P_2 and P_3 the probabilities of reaction R_1, R_2 and $\overline{R}_1 \cdot \overline{R}_2$ respectively, for the case $S_1 - S_2 > h$. Those probabilities are given by expressions developed by H. D. Landahl (1937), and are functions of $S_1 - S_2$. Similarly, denote by P'_1, P'_2 and P'_3 the respective probabilities of R_1, R_2 and $\overline{R}_1 \cdot \overline{R}_2$, when $S_2 - S_1 > h$, and denote by P''_1, P''_2 and P''_3 the same probabilities for $|S_2 - S_1| \leqslant h$. Now if we denote by p_1, p_2 and p_3 the probabilities respectively of R_1, R_2, and $\overline{R}_1 \cdot \overline{R}_2$, when $S_1 - S_2$ has any of the three possible values, and if we remember that inequalities $S_1 - S_2 > h$, $S_2 - S_1 > h$, and $|S_1 - S_2| \leqslant h$ are equally probable, then we find

$$\begin{aligned} p_1 &= \tfrac{1}{3}(P_1 + P'_1 + P''_1); \\ p_2 &= \tfrac{1}{3}(P_2 + P'_2 + P''_2); \\ p_3 &= \tfrac{1}{3}(P_3 + P'_3 + P''_3). \end{aligned} \tag{2}$$

The information per signal, or entropy H of the system, is

$$H = -(p_1 \log p_1 + p_2 \log p_2 + p_3 \log p_3), \tag{3}$$

and is a function of the chosen three pairs (S_1, S_2).

This result may be generalized by considering the probability $Q(\Delta)\,d\Delta$ that the difference $\Delta = S_1 - S_2$ will lie in the interval between Δ and $\Delta + d\Delta$. It is possible to find such a function $Q(\Delta)$ that the probabilities of inequalities (1) will be all equal. This requires that

$$\int_{-\infty}^{-h} Q(\Delta)\, d\Delta = \int_{-h}^{+h} Q(\Delta)\, d\Delta = \int_{+h}^{+\infty} Q(\Delta)\, d\Delta\,, \tag{4}$$

and there is an infinite number of functions $Q(\Delta)$ which satisfies that requirement. We now denote by $P_1(\Delta)$, $P_2(\Delta)$, and $P_3(\Delta)$ the probabilities of R_1, R_2, and $\overline{R}_1 \cdot \overline{R}_2$ respectively, when Δ has a given value. Instead of (2) we now have

$$\begin{aligned} p_1 &= \int_{-\infty}^{+\infty} P_1(\Delta)\, Q(\Delta)\, d\Delta; \\ p_2 &= \int_{-\infty}^{+\infty} P_2(\Delta)\, Q(\Delta)\, d\Delta; \\ p_3 &= \int_{-\infty}^{+\infty} P_3(\Delta)\, Q(\Delta)\, d\Delta\,. \end{aligned} \tag{5}$$

The entropy is again given by expression (3) and depends on the distribution function $Q(\Delta)$.

A cross-excitation between parallel neuron chains also acts to decrease the information per signal, by reducing the independence of choice of the individual signals.

Voluntary motor reactions are mediated mostly through the pyramidal tract, and partly through extrapyramidal fibers. The total number of fibers in the pyramidal tract is estimated at about 10^5. If they are all independent, this would give a possible maximum channel capacity for the pyramidal tract of about 10^7 bits sec^{-1}. Whether a reaction is caused by an external or internal stimulus, in either case the pyramidal cells constitute one of the last steps in transmission. The transmission from stimulus, whether external or internal, is done through some as yet unknown region of the brain. Since neither the number of cells involved, nor their connections is known, the channel capacity cannot even be guessed. We shall denote that channel capacity by C^*.

Now consider any set of motor reactions. Each reaction may be considered as a signal. If they are independent, and if there are n such reactions, the information per signal is equal to $\log_2 n$ bits. In general, the different signals are not independent, and this reduces the information H per signal. Thus, for example, a flexor and a corresponding extensor cannot contract simultaneously. If in a reaction pattern all reactions are completely independent, we may characterize such a pattern as physiologically completely uncoordinated. No prediction can be made about a subsequent signal from

the knowledge of preceding signals. If a certain degree of coordination is introduced, then certain more or less fixed patterns appear. By observing the beginning of a coordinated movement it is possible to predict with considerable probability its future course, at least up to a point. Physiologically the coordination is due to neural connections at higher neural levels. It reduces the number of possible reactions. From the point of view of the communication theory coordination reduces the entropy of the reaction pattern by reducing the independence of each reaction. The more coordinated or rigid a reaction pattern, the less information it can convey per signal. If H^* denotes the entropy per signal of a reaction pattern, and H_m the maximum entropy per signal which the pattern could have, if completely uncoordinated, then we may define with C. Shannon the quantity

$$r = 1 - \frac{H^*}{H_m} \tag{6}$$

as the redundancy of the reaction pattern. From what has been said above, it is natural to consider the redundancy r of a motor reaction pattern as a measure of the degree of its coordination. It is difficult to see at present how the redundancy can be measured for some very general types of motor patterns. It can, however, be measured for a very important type of patterns, namely, speech. (Shannon and Weaver, 1949). This possibility opens the way of verifying some interesting conclusions.

If C^* is the capacity of the central channel in bits sec^{-1}, and H^* is the entropy of the reaction in bits $signal^{-1}$, then the maximum rate of transmission T_{max} of *signals* is

$$T_{max} = \frac{C^*}{H^*} \text{ signals } sec^{-1}. \tag{7}$$

The average time $\bar{t}$ between the signals would be given in that case by H^*/C^*, if the speed of the reaction were limited only by the speed of transmission. Actually there are some purely physical limitations to the speed of muscular reactions. Those limitations set a lower limit τ to the average time between signals. Hence, with a better approximation, we have:

$$\bar{t} = \tau + \frac{H^*}{C^*}. \tag{8}$$

The maximum actual rate of performance of reactions per second is, therefore,

$$U_m = \frac{1}{\bar{t}} = \frac{C^*}{\tau C^* + H^*}. \tag{9}$$

Solving equation (6) for H^* and introducing the solution into equation (9) gives

$$U_m = \frac{C^*}{\tau C^* + H_m(1-r)}. \tag{10}$$

When applied to speech, expression (10) establishes a relation between the measurable quantities U_m, H_m, and r of a language. The greater the redundancy of a language the faster it can be spoken. Expression (10) could be best verified on polylingual individuals, using the same individual for several languages, and then measuring the constancy of C^* and τ.

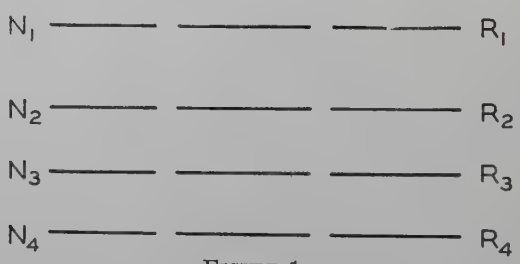

FIGURE 1a

The neurophysiological basis of this phenomenon is clear. If the reactions R_1, R_2, R_3, R_4 in Figure 1a are mediated by independent chains, then it requires the successive firing of the 4 central neurons, N_1, N_2, N_3, and N_4 to produce the sequence R_1, R_2, R_3, R_4. If, on the other hand, due to cross-connections, the firing of a single neuron N_1 results in the sequence R_1, R_2, R_3, R_4 (Figure 1b), then

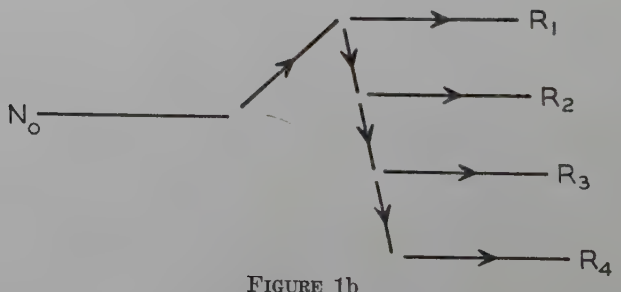

FIGURE 1b

an economy of time will be effected. Cross connections of that type may be viewed as a mechanism for coding. The firing of N_0 may be

considered as a code signal for the sequence R_1, R_2, R_3, R_4. The cross connections may be either fixed and innate, or they may develop as a result of conditioning. If a sequence of initially independent reactions occurs sufficiently frequently, central cross connections of a conditioned nature (Rashevsky, 1948) will develop, and this will result both in an economy of central neurons necessary to produce the sequence and in an economy of time. Such an encoding has apparently developed in the English, and probably in other, languages in the course of time, as evidenced by K. Zipf's findings (1935) that the shorter a syllable the greater the frequency of its occurrence. Any process of learning a complex motion pattern results in such a conditioning between the different elements of the pattern and in a resulting encoding and increase in rate of performance. The first attempt to perform a new complex pattern results in slow performances. With learning, the speed increases.

If we concede that thought is merely a covert, "unspoken" speech (Jacobson, 1931), then equation (10) may perhaps be applied to the speed of abstract thinking. Solution of an abstract nonmathematical problem may take less time if the individual thinks in a language with greater redundancy. Whether this is so or not future experimentation may show. The theory proposed here definitely suggests quantitative experiments along new lines.

While the more redundant a language, the more rapidly it may be spoken and perhaps thought, yet the less information it carries per signal. If, as suggested elsewhere (Rashevsky, 1948, chapter xlii), the rational solution of a problem requires a transfer of as much information as possible from one field to another, then a redundant language may be a handicap. If the *abstract* language is not sufficient to carry all the desired information, the individual may supplement it with other motor reactions, such as are involved in visualization (Jacobson, 1930). It has been noticed sometimes that English speaking scientists use considerably more visual "models" in their thinking than, for example, German scientists. Typical in the use of "visual models" are, for example, Lord Kelvin and Maxwell, as compared, for instance, to Plank or Einstein. If the redundancy of German is less than that of English, the above situation may be understood. Other cultural factors undoubtedly also play an important part. It may, however, be possible to establish a positive correlation between the redundancy of a language and the tendency to think in visual terms. This, of course, will not mean that the choice of the language is the cause of the thinking habit or vice versa. They both may be results of some common factor.

Now let an individual face a situation consisting of n possible mutually exclusive events, concerning the occurrence of which he does not know anything, and which therefore are equally probable to him. Using the terminology of the communication theory, we may say that in this case the amount of information which the individual can obtain from the situation is $\log_2 n$ per event, or $n \log_2 n$ for the whole situation. Gradually, as a result of observation or thinking or both, the individual acquires some knowledge about the events. He finds out that the probability of occurrence of some of them is $p_i > 1/n$; the probability of occurrence of others is less than $1/n$. His knowledge of the situation has increased. As a measure K of this knowledge we may conveniently take the expression

$$K = \tfrac{1}{2} \sum_{i,j} (p_i - p_j)^2. \tag{11}$$

When all events are equally probable, $K = 0$, as should be the case. When one event, the rth, is known with certainty, then $p_r = 1$; $p_i = 0$ $(i \neq r)$, and $K = 1$. But as K increases, the information which the individual can obtain from the situation decreases. It is now equal to

$$H = -\sum_i p_i \log_2 p_i \tag{12}$$

per event, or

$$I = nH = -n \sum_i p_i \log_2 p_i \tag{13}$$

for the whole situation. The more an individual knows about a finite situation, the less information he can extract from that situation. The statement sounds trivial, but the above equations give it a quantitative formulation.

Instead of expression (11) we may take as a measure of knowledge the difference between the maximum entropy, $\log_2 n$, and the actual entropy H as given by (12). Thus we may put

$$K = \log_2 n - H = \log_2 n + \sum_i p_i \log_2 p_i. \tag{14}$$

Since H decreases when K increases, therefore an individual who strives for a large knowledge and yet also enjoys the process of acquiring information will have to compromise somewhere between maximum knowledge and maximum available information. If he maximizes K, then he will know with certainty one event, say the rth $(p_r = 1)$. Maximizing H he choses to know nothing about every event. He may choose to maximize the product KH. If K is given

by (11) then this amount to maximizing the expression

$$L = -\tfrac{1}{2} \sum_{ij} (p_i - p_j)^2 \sum_i p_i \log p_i . \tag{15}$$

When all $p_i = 1/n$, $L = 0$; it is also equal to zero for $p_r = 1$; $p_i = 0$ $(i \neq r)$, and it is positive everywhere else. Hence there is at least one set of values $p_1^*, p_2^*, \cdots, p_n^*$, for which L has a maximum. If we choose as measure of K expression (14), then

$$L = KH = (\log_2 n - H) H . \tag{16}$$

This has a maximum for

$$H = \log_2 \sqrt{n} , \tag{17}$$

or

$$-\sum_i p_i \log p_i = \log_2 \sqrt{n} . \tag{18}$$

Equation (18) determines a hypersurface in an $(n - 1)$-dimensional space. All points in that hypersurface have coordinates p_i which maximize L.

To allow for individual preferences for either K or H we may maximize the quantity

$$M = K^\alpha H^\beta \tag{19}$$

where α and β are individual parameters, both greater than unity. Using expression (11) we may maximize a linear combination of K and H. This is impossible if we choose expression (14).

We may consider the same problem from a different, perhaps somewhat more realistic, angle. Observation may show that the n events are neither all mutually exclusive, nor completely independent. Thus we may observe that events a_i and a_k sometimes occur separately, sometimes together. This results in a formation of an association between a_i and a_k in our minds. If a_i and a_k always occur together, then this actually reduces the number of independent events from n to $n - 1$. Moreover, if the probability of a_i and a_k remains $1/n$, then in the new situation of $n - 1$ events there are $n - 2$ events a_r $(r \neq i, k)$ with a probability $1/n$, and one event $a_i + a_k$ with the probability $2/n$. This makes the entropy of the situation per event less than $\log_2 (n - 1)$.

Quite generally, the problem may be treated in the following way.

Let the probability of a_i, whether alone or in combination with a_k, be $1/n$. Let the same hold for the probability of a_k. Let $p_{ik'}$

denote the probability that a_k appears when it is known that either a_i has appeared alone or both have appeared. Let p_{ki}' denote the probability that a_i appears when it is known that either a_k has appeared alone or both have appeared. In general we may have $p_{ik}' \gtrless p_{ki}'$. If p_i', respectively p_k', denotes the probability that a_i, respectively a_k, appears alone when it is known that it has appeared either alone or in combination, then

$$p_i' + p_{ik}' = 1; \quad p_k' + p_{ki}' = 1. \tag{20}$$

Since the probability of a_i appearing either alone or in combination, when nothing is known about it, is $1/n$, while the probability of its appearing alone, when it is known that it has appeared either alone or in combination, is p_i', therefore probability p_i that a_i will appear alone in the whole situation is

$$p_i = \frac{p_i'}{n} = \frac{1 - p_{ik}'}{n}. \tag{21}$$

Similarly

$$p_k = \frac{p_k'}{n} = \frac{1 - p_{ki}'}{n}. \tag{22}$$

Finally the probability p_{i+k} that a_i and a_k appear together when it is not known that either one of them has appeared is given by

$$p_{i+k} = \frac{p'_{ik} + p'_{ki}}{n}. \tag{23}$$

The situation now consists of $n + 1$ possible events:

$$a_1, a_2, \cdots a_n; a_i + a_k. \tag{24}$$

Of those $n + 1$ events $n - 2$ events $a_r (r \neq i,k)$ have the probability $1/n$; the event a_i has the probability p_i as given by (21); the event a_k has the probability p_k as given by (22), and the event $a_i + a_k$ has the probability $(p'_{ik} + p'_{ki})/n$. Hence the entropy is equal to

$$\begin{aligned} H = {} & \frac{n-2}{n} \log_2 n - \frac{1 - p'_{ik}}{n} \log_2 \frac{1 - p'_{ik}}{n} \\ & - \frac{1 - p'_{ki}}{n} \log_2 \frac{1 - p'_{ki}}{n} - \frac{p'_{ik} + p'_{ki}}{n} \log_2 \frac{p'_{ik} + p'_{ki}}{n}. \end{aligned} \tag{25}$$

When $p'_{ik} = p'_{ki} = 0$, this reduces to

$$H = \frac{n-2}{n} \log_2 n + \frac{2}{n} \log_2 n = \log_2 n , \tag{26}$$

as should be the case, for we then have n equally probable independent events. If $p'_{ik} = p'_{ki} = 1$, then

$$H = \log_2 n - \frac{2}{n} < \log_2 (n-1) . \tag{27}$$

Thus if we know with certainty ($p'_{ik} = p'_{ki} = 1$) that two (or more) events are associated with each other, we decrease the entropy of the situation. If our knowledge is not absolute ($p'_{ik} < 1$; $p'_{ki} < 1$) then the entropy is reduced by a lesser amount. We can again attempt to define a proper measure K of knowledge and to construct such a function of K and H that its maximum will determine the individual's choice of an optimum between greatest knowledge and greatest possible information to be obtained.

It is an interesting and important problem to determine what function of K and H an individual actually tries to maximize and how K is expressed in terms of the p_i's, because this determines in a sense the individual's "philosophy." Maximizing K will result in a certain knowledge of one particular constellation: the rth event occurs always, other events never occur. But this precludes any further discussion of any other constellation in the situation. On the other hand, when H is near its maximum, the individual's knowledge of the single constellation is small, but the number of possible constellations is large. We have here, in a sense, not only a "mathematical formulation," but also an actual refinement of the joke which defines the specialist as one who knows everything about nothing, and the philosopher or diletante as one who knows nothing about everything. We see that the specialist must be defined as one who knows everything about only one thing. Perhaps the values of the p_i's which maximize either expression (15) or (16) may be considered as corresponding to the behavior of the "ideal" scientist, who knows enough about a sufficiently large number of things, but who is never absolutely certain of anything, and therefore is willing to question everything he knows, and thus avoid dogmatism. The contemporary shift from the old single-valued Aristotelian orientation to the multivalued probabilistic non-Aristotelian orientation illustrates the point. In a sense, the non-Aristotelian orientation may be justified by the further development of the theory outlined here.

The preference for either a larger K or a larger H may be de-

termined not solely by personal predilections, but may be to some extent imposed upon us by external conditions. A practical physician knows that drug X is helpful for a particular dangerous disease. Though the beneficial results are not always obtained, they are observed in such a large percentage of cases that expediency definitely dictates the use of X for that disease. The physician will instruct a younger doctor in no uncertain terms—"If the disease occurs, use X." But, with this practical attitude, there is hardly any possibility left for discovering that another drug, Y, may be better in some cases than X. On the other hand, the scientist or clinician, while knowing that X is *practically* the best remedy for the disease, will keep in mind that this knowledge is not certain. He will try other drugs on his patients, and is very likely to lose more patients in this process than the practical physician will with the use of X only. But in the long run the clinician has a chance to discover the more efficient drug Y. In other words, he can acquire more information by his method of approach which involves uncertainty.

An intellectual individual learns as long as he lives. It is natural to assume that the rate of increase of his knowledge K is the greater, the greater the available information. If we consider a linear relation, we may write, with κ as a constant,

$$\frac{dK}{dt} = \kappa H. \tag{28}$$

If for K we use expression (14), equation (28) becomes

$$\frac{dK}{dt} = \kappa(\log_2 n - K). \tag{29}$$

Integrated with the initial condition that for $t = 0$ (at birth), $K = 0$, equation (29) gives

$$K = (1 - e^{-\kappa t}) \log_2 n \tag{30}$$

and

$$\frac{dK}{dt} = \kappa e^{-\kappa t} \log_2 n. \tag{31}$$

Since t in this case is proportional to the age of the individual, we see that the rate of learning decreases with age.

The maximum amount of knowledge is given, according to (30), by $\log_2 n$. The number n, while a characteristic of the environment, is nevertheless to some extent determined by the individual. The

larger the scope of interest of an individual, the larger the number n of events to which he gives his attention. If n is very large the individual can "afford" fairly precise knowledge of some events without impairing materially the amount of information available to him. The quantity $\log_2 n$ may be considered as the measure of the individual's "span of interest." The larger the "span of interest" of an individual, the larger will be his rate of learning at a given age.

II. *The Social Group.*

Consider a sufficiently large group of N_0 individuals which is characterized by properties discussed previously (Rashevsky, 1950, chap. xii). That is, each individual *always* exhibits *one* of two mutually exclusive reactions, R_1 and R_2, and each has a bias ϕ in favor of one or the other of the two reactions. The probabilities P_1 and P_2 of R_1 and R_2 respectively are functions of ϕ. Assuming with H. G. Landau (1950) a normal distribution of the random fluctuations with a standard deviation σ_1 and putting

$$G\left(\frac{x}{\sigma}\right)=\frac{1}{\sqrt{2\pi}\,\sigma_1}\int_{-\infty}^{x} e^{-\frac{x^2}{2\sigma_1^2}}\,dx, \tag{32}$$

we have:

$$P_1=G\left(\frac{\phi}{\sigma_1}\right);\quad P_2=1-G\left(\frac{\phi}{\sigma_1}\right). \tag{33}$$

For $\phi=0$; $P_1=P_2=\frac{1}{2}$.

We consider furthermore that the reaction R_1 produced by another individual acts as a stimulus favoring R_1 in a given individual, while a reaction R_2 of another individual favors R_2 in a given one. We shall neglect here the variations in the intensities of the reactions, and consider both R_1 and R_2 as always of the same intensity S. Denoting by P' the probability of reaction R_1, when the other individual exhibits that reaction too, and choosing units for S properly we now have

$$P'=G\left(\frac{\phi=S}{\sigma_1}\right). \tag{34}$$

The probability P'' of R_1 when the other individual exhibits R_2, is given by

$$P''=G\left(\frac{\phi-S}{\sigma_1}\right). \tag{35}$$

As we have seen at the beginning of this paper, such an individual may be considered as capable of transmitting a maximum of one bit of information per reaction. Suppose we have a linear chain of n such individuals. How much information can be transmitted per signal through such a chain?

To answer this question, let us at first consider the very special case in which the biases ϕ of all the n individuals are the same. Denote by p_n the probability of the reaction R_1 by the nth individual when it is known that the zeroth individual has exhibited the reaction R_1. The probability p_n is the sum of the probabilities that R_1 is exhibited when the $(n-1)$st individual exhibits R_1, and of the probability that R_1 is exhibited when the $(n-1)$st individual exhibits R_2. Since the probabilities of the $(n-1)$st individual exhibiting R_1 and R_2 are respectively p_{n-1} and $1-p_{n-1}$, we have

$$p_n = p_{n-1} P' + (1-p_{n-1}) P'', \tag{36}$$

or

$$p_n - (P' - P'')\, p_{n-1} = P''. \tag{37}$$

This is a difference equation with constant coefficients. We seek the value p_n for the case in which $p_0 = 1$. Putting

$$P' - P'' = A; \quad P'' = B, \tag{38}$$

the difference equation (37) becomes

$$p_n - A\, p_{n-1} = B. \tag{39}$$

Its solution, for $p_0 = 1$, is

$$p_n = A^n + B\frac{1-A^n}{1-A}. \tag{40}$$

We may similarly calculate the probability q_n of a reaction R_2 by the nth individual, when the zeroth has exhibited R_1. Denote by Q' the probability of the nth individual exhibiting R_2, when the $(n-1)$st exhibits R_2, and by Q'' the probability of the nth individual exhibiting R_2, when the $(n-1)$st exhibits R_1. We have

$$Q' = 1 - P''; \quad Q'' = 1 - P'. \tag{41}$$

In a similar manner as that by which we arrived at (37) we now have

$$q_n = (Q' - Q'')\, q_{n-1} = Q''. \tag{42}$$

The initial condition is now, however, $q_0 = 0$.

Hence, putting

$$Q' - Q'' = A'; \quad Q'' = B', \tag{43}$$

we have:

$$q_n = B' \frac{1 - A'^n}{1 - A'}. \tag{44}$$

Remembering (38) and (41) we find from (40) and (44), after rearrangements, that

$$p_n + q_n = 1, \tag{45}$$

as should be the case.

Expression (40) is a particular form of the more general expression

$$p_n = p_0 A^n + B \frac{1 - A^n}{1 - A} \tag{46}$$

which holds when $p_0 > 0$. We thus see that the first term of (40) depends in general on the initial reaction, the second is a characteristic of the "channel" only. As n tends to infinity, the first term of (40) tends to zero, because $A < 1$, the second to

$$p_{c\infty} = \frac{B}{1 - A} = \frac{P''}{1 - P' + P''} < 1. \tag{47}$$

Thus the probability of a reaction becomes, for an infinite value of n independent of the initial reaction or signal. No information is then transmitted at all. We may naturally interpret the first term of (40) as representing the probability of R_1 due to transmitted information, while the second term of (40) may be said to represent the probability of R_1 due to "channel noise." The effect of the channel noise increases from zero to the constant asymptotic value $p_{c\infty}$ given by (47).

If the bias ϕ is zero, then, as seen from expressions (34) and (35), $P' + P'' = 1$ and $p_{c\infty} = \frac{1}{2}$, a result which is intuitively evident.

Putting

$$p_{in} = A^n; \quad p_{cn} = \frac{B(1 - A^n)}{1 - A}, \tag{48}$$

we may write expression (40) as follows

$$p_n = p_{in} + p_{cn} . \tag{49}$$

The probability p_{cn} due to channel noise may in principle be calculated or determined empirically. When $p_0 = 1$, $q_0 = 0$, then the probability of q_n, given by equation (44), is due to channel noise only. If the bias ϕ is zero, then, as we have seen above, $P' + P'' = 1$. Together with (41) this gives $P'' = Q''$. Hence, because of equations (38) and (43) we have $B = B'$. From (38) and (41) we also find $A = A'$. It follows that for $\phi = 0$, $q_{cn} = p_{cn}$, as should be the case. Regardless of the channel length n, the contribution of the channel noise to R_1 and R_2 is equally probable for $\phi = 0$. Suppose we receive at the output end M signals, of which we shall have Mp_n signals R_1. Of those Mp_n signals R_1 a number Mp_{cn} is due to the channel noise and Mp_{in} is due to the signal R_1 at the input. For each of the Mp_{cn} signals the *a posteriori* probability of R_1 at the origin is $\frac{1}{2}$. For each of the Mp_{in} signals this *a posteriori* probability is 1. Hence the average *a posteriori* probability of R_1 at the origin when R_1 is observed at the end is

$$\tilde{p}_n = \frac{\frac{1}{2}Mp_{cn} + Mp_{in}}{Mp_{cn} + Mp_{in}} = \frac{p_{cn} + 2p_{in}}{2p_n} . \tag{50}$$

As n increases, p_{in} tends to zero, while p_{cn} tends to p_n. Hence the *a posteriori* probability $\tilde{p}_n$ tends to $\frac{1}{2}$.

The transmitted information per signal at the end of the channel when at the origin it is equal to 1, is given according to C. Shannon (1949), by

$$R_n = 1 - \{ -\tilde{p}_n \log_2 \tilde{p}_n - (1 - \tilde{p}_n) \log_2 (1 - \tilde{p}_n) \} . \tag{51}$$

As n increases and $\tilde{p}_n$ tends to $\frac{1}{2}$, the expression in braces tends to 1, and R_n tends to zero. Introducing (34) and (35) into (38), then the latter into (48), then introducing (48) into (50) and finally the latter into (51), we obtain R_n as a function of the length n of the chain.

The above situation is, however, complicated by the fact that the values P' and P'' are in general different for different members of the chain, because different individuals have different biases ϕ. In general instead of equation (39) we shall have

$$p_n - A_n p_{n-1} = B_n . \tag{52}$$

Denoting by ϕ_n the bias of the nth individual, we have:

$$A_n = G\left(\frac{\phi_n + S}{\sigma_1}\right) - G\left(\frac{\phi_n - S}{\sigma_1}\right),$$
$$B_n = G\left(\frac{\phi_n - S}{\sigma_1}\right). \tag{53}$$

If A_n and B_n are definite known functions of n, then the general solution of (52) for $p_0 = 1$, is

$$p_n = A_1 \cdots A_n \left(1 + \sum_1^n \frac{B_m}{A_1 A_2 \cdots A_m}\right), \tag{54}$$

which reduces to (39) when $A_n = A$; $B_n = B$. Unfortunately the A_n's and B_n's are not known functions of n, because ϕ varies at random from individual to individual. The simplest thing to do is to use equations (39) and (40) with constant coefficients, but introduce for A and B appropriate average values $\bar{A}$ and $\bar{B}$. If $N(\phi)$ denotes the distribution function of ϕ we may put

$$\bar{A} = \int_{-\infty}^{+\infty} \bar{A}(\phi)\, N(\phi)\, d\phi; \quad B = \int_{-\infty}^{+\infty} B(\phi)\, N(\phi)\, d\phi, \tag{55}$$

where

$$A(\phi) = G\left(\frac{\phi + S}{\sigma_1}\right) - G\left(\frac{\phi - S}{\sigma_1}\right);$$
$$B(\phi) = G\left(\frac{\phi - S}{\sigma_1}\right). \tag{56}$$

We thus see, that the transmission of information along a "social chain" depends on the distribution function $N(\phi)$.

A somewhat more rigorous, but elaborate, discussion of this problem will be given in a subsequent paper.

If the population density is sufficiently high, we may introduce into (40) the variable

$$x = an \tag{57}$$

where a is the average distance between two adjacent individuals, and x the actual physical length of the chain or the distance between its two ends. Then R_n becomes a function $R(x)$ of x, and the equation developed above gives us the amount of information transmitted through a chain of individuals as a function of the physical length

of that chain.

We now have instead of (51)

$$R(x) = 1 + \tilde{p}(x) \log \tilde{p}(x) + [1 - p(x)] \log_2 [1 - p(x)]. \qquad (58)$$

Now we may compute the amount of information per signal that can be transmitted from one individual to another, when the distance between the individuals is equal to r and when they both are members of a social group of constant population density. In such a case there is a large number of possible channels between the two individuals, through which the information may be transmitted. There exists a shortest channel, whose length is close to r. But there are many others, much longer ones, such as channels I, II and III on Figure 2. Some channels, as II and III, may have general links in

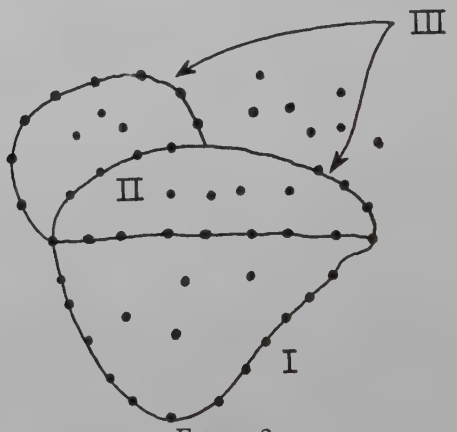

FIGURE 2

common. All these possible channels are connected in parallel, and therefore the total information transmitted through them is the sum of the information transmitted through each channel separately. The evaluation of the total number of possible discrete channels is a rather complex problem. We may, however, use a good approximation, by passing to a continuous case. Consider the transmission of information between individuals 1 and 2 (Figure 3). Let $ds = r'dr'd\theta$ be an element of area at the point 3, which lies on a circle of radius r'. If δ is the density of population, then the element ds contains $\delta r'dr'd\theta$ individuals. From the individual 1 to any one of the above individuals there is a shortest chain of length r'. And similarly from each of those individuals to the individual 2 there is a shortest chain of length r''. Hence, there are altogether $\delta r'dr'd\theta$

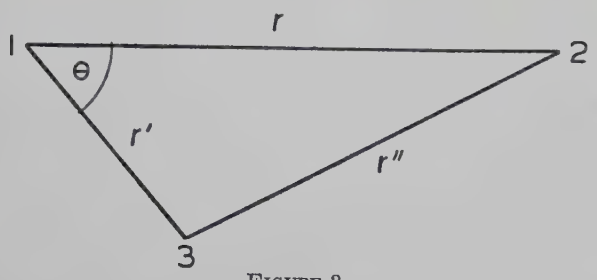

FIGURE 3

chains of length $r' + r''$ between individuals 1 and 2, going through point 3. The amount of information transmitted through those chains is

$$dI = R(r' + r'')\ \delta r' dr' d\theta\ , \tag{59}$$

where $R(r' + r'')$ is given by (58), in which we put $x = r' + r''$.

But

$$r'' = \sqrt{r^2 + r'^2 - 2rr' \cos\theta}\ . \tag{60}$$

Hence

$$dI = \delta\, R(r' + \sqrt{r^2 + r'^2 - 2rr' \cos\theta})\ r' dr' d\theta\ . \tag{61}$$

The total information transmitted from 1 to 2 is obtained by integrating expression (61) with respect to r' and θ over the whole possible range of those two variables. The variables r' and θ integrate out and the total information becomes a function of r only:

$$I(r) = \delta \int_{r'} \int_{\theta} R(r' + \sqrt{r^2 + r'^2 - 2rr' \cos\theta})\ r' dr' d\theta\ . \tag{62}$$

The limits of integration in (62) will in general depend on the size and shape of the area occupied by the population. This is not surprising, since the number and lengths of possible channels depend on the size and shape of the area. Since $R(x)$ decreases to zero exponentially, as seen from equations (40), (51) and (57), therefore when the two individuals are not too close to the periphery, and when their distance is small compared to the size of the area occupied by the social group, $I(x)$ will be practically independent on the size and shape of the area. It is even possible that the double integral may have a meaning for an infinite population of uniform density.

It is readily seen that an individual receives more information when he is located in the center of a two dimensional area, than when

he is a member of a linearly arranged chain of individuals.

In a previous study (Rashevsky, 1950) effects of distance between individuals on their tendency to imitate each other were studied. It is natural to assume that the imitation effect will be proportional to the amount of information received. In that case the expression $I(r)$ may well be used for the kernel $K(r)$ introduced in the above mentioned study.

The next step in the present study would be to generalize our results to the case of variable intensity of reactions R_1 and R_2, as well as to introduce thresholds, somewhat in the manner as has been done in connection with equations (5). The most important generalization, however, would be toward more than two mutually exclusive, or non-exclusive reactions.

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INDEX TO VOLUME 12

INDEX OF AUTHORS

INDEX OF SUBJECTS

BOOK REVIEW